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EXPLANATION OF DRAWINGS.

TABLE I.

FIG. 1.—*Ossifying transverse septum behind ciliary body and lens.—i*, iris; *p*, ciliary processes; *m*, ciliary muscle; *o*, lens; *a*, *b*, ossified portions of septum; *d*, fibrous portion of septum; *c*, post. capsule; *r*, retina.

FIG. 2.—*Formation of ossifying false membrane* (f, f_1, f_2), *starting from the capillary layer of the choroid* (*c*, *c*), *in the region of the ora serrata, and stretching transversely through the vitreous.—m*, osteoid tissue in septum; *b*, blood-vessels in septum; *v*, blood-vessels in choroid; *e*, pigment, and *h*, hyaline membrane of choroid.

FIG. 3.—*Osteoid tissue* (*a*) *in pseudomembrane* (*f*) *with vascular* (*v*) *medullary spaces* (*h*).—*o*, *o*, line of ossification; *m* circumscribed clusters of chalk; *c*, lens capsule; *d*, fibrous covering.

FIG. 4.—*Osseous plate* (*p*) *in capillary layer of choroid, around optic nerve entrance.—ir*, iris; *c*, cil. body; *l*, disorganized lens; *m*, transverse septum drawn forward.

FIG. 5.—*Formation of bone* (*o*) *in chorio-capillaris* (*n*).—*m*, nutrient canals; *v*, blood-vessel; *c*, colloid globules upon hyaline membrane of choroid.

FORMATION OF BONE IN THE EYE.

By H. KNAPP.

(Plate I.)

Formation of true bone within the eyeball is not of so rare an occurrence that a mere statement or short description of its existence, in one or several cases, could be considered an addition to the pathology of the eye. Nevertheless this subject is far from being sufficiently elucidated. For some time all very hard productions were believed to be bony structures; afterwards the contrary opinion prevailed, which denied altogether the formation of true bone in the eye, admitting only the occurrence of chalky concretions. At present the existence of both chalky deposits and real osseous tissue, which are often found simultaneously in the same case, is demonstrated beyond a doubt. Future investigations, therefore, have other questions to solve, some of which I beg leave to indicate.

1. *Which of the membranes, and which layer of each, are the starting-points of ossification?*

V. Wittich (Virch. Archiv V., p. 580) described formation of bone in the vitreous, but it remains doubtful

in which part it began. *Förster* (Atlas der Micr.-Path. Anatomie, Taf. XXXV.) found a hard tumor, the size and shape of the *crystalline lens*, occupying the place of the latter, to consist of real bone, with Haversian canals and a concentric arrangement of the bone corpuscles around them. He says that the specimen was evidently no ossification of the lens; but he leaves it uncertain whether, in his opinion, the bone had developed in the vitreous body or sprung from new-formed connective tissue that had replaced the destroyed lens.

Formation of bone within the lens has been mentioned, but the observations want confirmation, and are doubted by such authorities as Profs. *Virchow* and *H. Müller*. I think the possibility of ossification in the lens cannot absolutely be denied, but its occurrence must certainly be very rare. It is incontestable that formation of connective tissue within the lens may take place. Chalky or cretaceous cataract is known to every oculist from personal experience. Since connective tissue is capable of ossifying wherever it exists, it may do so in the lens. *Petrification* is a process of *regressive* metamorphosis, and therefore not infrequent in old cataracts, especially those of a degenerative kind. *Ossification*, on the contrary, is an *active* process, requiring a good supply of nutritive material, for which the crystalline lens offers very unfavorable conditions, possessing in its normal state no blood-vessels. The *retina* and *vitreous* body are not very apt either to become the origin of ossification, the latter having no vessels of its own, and

the former also being but scantily supplied with blood. The vascular net of the retina, dependent on but one minute arterial trunk, and a very few insignificant anastomoses with the small posterior ciliary arteries, ramifies only in the inner layers of the retina. This makes it plain why formation of bone has not yet been really demonstrated, either in the retina or in the vitreous body, whereas earthy deposits in them are not infrequent.

The *uveal tract* is the only coat in the eye which can furnish abundant supply of nutritive material to any new formation. It is, therefore, but natural that products of various kind have been found to originate in it. New-formed osseous tissue was always observed on its inner surface, and the blood-vessels of the bone arose from those of the choroid. Among all portions of the uveal tract its *capillary layer* is the principal source of nutrition, and ossification seems, indeed, to originate always either in the same, or in inflammatory products derived from it.

2. *What diseases are most likely to lead to ossification?* All reported cases were of long duration, and exhibited plastic deposits coating the inner surface of the choroid, and frequently extending along the ciliary processes and the posterior surface of the lens. In the excellent treatise of Stellwag "On the Diseases of the Eye" we find ossification described among the results of Irido-choroiditis (2d Americ. Ed., p. 232).

3. With regard to *prognostical* and *therapeutical*

questions we have to account for the *great differences in the tolerance* of eyes affected with ossific products. What is the reason that some of them do not show any unpleasant symptoms at all, bearing even an artificial eye without the least irritation? What, on the contrary, keeps others always subject to inflammatory exacerbations, leading even to sympathetic affections of their fellows?

I will defer the discussion of these questions until I shall have described some cases.

The first is that of a healthy-looking girl, 17 years of age, who consulted me in July, 1868. She had suffered from eye diseases from her early childhood. The *left* eye had always been most affected, and lost its sight completely about three years previously. From that time up to the present moment it had been the seat of repeated painful inflammations, in which the *other eye* commonly participated to a less degree, showing great sensibility to the touch, intolerance of light, lachrymation, and dimness of sight, so that she was not able to read, or see anything clearly with it. This eye, however, recovered after each attack; but the patient said her sight of late was no longer so good as it used to be. She would prefer to have the other eye taken out, if that would preserve the good one.

On examining the eyes I found in the *right* $M = \frac{1}{8}$; $S = \frac{1}{4}$, and slight opacities in the peripheral part of the cornea, not sufficient to account for the stated impairment of visual power. The interior of the eye displayed nothing remarkable.

The *left eye* was shrunken, somewhat square-shaped by the traction and pressure of the recti muscles. The cornea was dim, anterior chamber shallow, iris discolored, its pupillary margin irregular and adherent to the anterior capsule, which itself was covered by a white membrane. The sclerotic had its usual color. The vessels branching

over it were somewhat increased in number and caliber. The sensibility of the cornea was undiminished. The two *posterior thirds of the globe*, as far as they could be brought to view, *were to the touch as hard as wood;* while the anterior third—that is, the whole cornea and the adjoining sclerotic in the breadth of six millimetres—offered a normal resistance to a finger or probe pressing upon it. The eye had no perception of light.

My opinion was that, in early youth, a scrofulous condition of the system had caused pustular ophthalmia and corneal specks, after which, in the left eye, irido-choroiditis had set in, which had run a chronic course, produced detachment of the retina and plastic effusions at the inner surface of the choroid, which afterwards had become ossified. In the lens and retina I expected to find degeneration into connective tissue and calcareous deposits. I assumed that the ciliary body and iris were still in a state of chronic inflammation inducing sympathetic difficulty in the other eye, which, in fact, became irritated and dim whenever an inflammatory exacerbation reappeared in its fellow. Fearing very strongly that it might sooner or later become seriously injured, evidences of beginning sympathetic suffering having, in reality, already manifested themselves, I thought it incumbent upon me to comply with the patient's own suggestion to remove the degenerated eye.

Enucleation was performed, and the wound healed readily. Soon after, the patient felt very comfortable, and had her sight in the right eye improved from $\frac{1}{4}$ to $\frac{1}{2}$ of the normal acuteness. She never had any obscurations or irritations after the operation, and wears an artificial eye without inconvenience.

Anatomical examination of the extirpated eye-ball. Immediately after the enucleation, I made a cursory inspection of the globe, postponing its thorough examination until it was hardened in alcohol.

The different diameters of the ball measured, the antero-posterior and horizontal, 18 millimetres each; the vertical, 17 *mm.* The cornea was likewise reduced in every direction, and was, together with the adjacent part of the sclerotic, soft to the touch; but the whole middle and posterior part of the sclerotic appeared hard as bone, and entirely unyielding when pressed upon. The *cut surface of the optic nerve* revealed nothing worth mentioning, except a considerable diminution of its area, a condition of simple atrophy. The globe was opened by a meridional section which could be performed in the anterior part with the knife, but required strong scissors to complete it through the equatorial and posterior region. A *small quantity of fluid*, slightly tinged with yellow, escaped, which, when examined under the microscope, showed but a few scattered heaps of pigment, fat granules, and epithelial cells, derived from the innermost choroidal layer.

The *crystalline* lens was in its normal position, of usual size, transparent in the centre, but studded with small *white, rough plaques* in both cortices, less in the anterior than in the posterior. They presented themselves under the microscope as clusters of granules, each being about the size of a white blood-corpuscle, remained unchanged in sulphuric ether, but dissolved perfectly in muriatic acid, producing a few air-bubbles only. According to these reactions, they were *chalky deposits*, with a preponderance of the phosphate over the carbonate of lime. In addition there was found in the crystalline a great

amount of well-preserved lens fibres, together with irregular fibrillæ having the appearance of connective tissue, besides fat in granules and clusters, dissolving in ether.

The *retina*, wholly degenerated into connective tissue, was stretched as a folded curtain through the vitreous chamber, being attached anteriorly to the ciliary body, and enclosing near its insertion into the optic nerve a yellowish, granular, earthy lump, the size of a large pea, consisting of phosphate of lime. A great many blood-vessels pervaded the fibrillated membrane, which was studded with fresh and old hemorrhages. The granules of chalk appeared, under the microscope, in part even and smooth, similar to oil globules, in part striped by concentric circles, like amyloid bodies. They were not found alone, but aggregated in smaller or larger clusters, the boundaries of which towards the surrounding tissue were distinctly defined.

From the region of the ora serrata arose a *grayish membrane*, ½ *mm.* in thickness, which stretched *transversely* through the vitreous body, parallel to the equatorial plane, its centre lying about 2 *mm.* behind the posterior pole of the lens.

The *sclerotic* appeared partially wrinkled, but normal in structure. *Its inner surface was invested by a threefold coat:* 1, by the outer layers of the choroid; 2, by a bony shell; and 3, by a fibrous tunic. The *lamina fusca* showed no remarkable deviations from its normal condition. The choroid, together with the adherent bony and fibrous capsule, could be detached, in one mass, from the apparently

normal sclerotic. The posterior part of the outer choroidal layer was rusty brown, while its anterior part was covered by a thin white mesh, continuous with the unchanged *ciliary ligament*. This white mesh showed, under the microscope, a dense interlacing of delicate fibres enveloping a quantity of small cells, viz.: white blood-corpuscles suspended in coagulated fibrine. The tunica vasculosa Halleri was in most places remarkably well preserved; in some, however, the vessels and stellate cells, pigmented as well as unpigmented, were diminished in size and number, and replaced by fibrillous connective tissue. The *whole capillary layer* of the choroid was absent and replaced by a *hard capsule*, extending from the optic disc to the pars plana of the ciliary body, and consisting of *true bone*, showing, under the microscope, not only perfect bone-corpuscles, but also in some places the concentric arrangement of the latter around the nutritive canals. This bony shell was lined at its inner surface by a soft *white layer*, consisting of delicate connective tissue fibres, arranged in bundles crossing one another at acute angles, and being abundantly interspersed with small round cells. This layer continued at the pars plana corporis ciliaris, where the bony shell ceased, into the thin membrane, stretching, like a septum, transversely through the anterior part of the vitreous. Under the microscope it revealed the structure of a false membrane. The *ciliary body* itself and the *iris* showed a low degree of simple atrophy without any degeneration. The *ciliary nerves* ran *intact* through the lamina fusca, and entered the ciliary muscle and iris as usual.

The remarkable feature of this case was the *limitation of the ossification to the capillary layer of the choroid.* A chronic plastic inflammation resulted in the metamorphosis of this layer into dense connective tissue, which subsequently was converted into real bone. The ossification began on the outer surface of the degenerated chorio-capillaris, bordering immediately on Haller's layer, without extending into it. The formation of new bone was evidently still going on at the inner surface of the bony shell, where the adjacent strata of the fibrous covering were in process of being converted directly into osseous tissue. No trace of the interposition of *cartilage* between the bone and its fibrous covering was visible, but in many places the transitional state between connective tissue and bone, the *osteoid* tissue, was well marked. This continuance of the fibrous covering on the inner surface of the bony shell into a transverse membranous septum behind the ciliary body and lens is very important. This septum, if the case had been allowed to run its course, would have also ossified, as is demonstrated by the following observations.

The opportunity to examine another specimen of ossification was given me by my friend Dr. H. Althof, of New York, who kindly added to the obligation by furnishing me with the following history of the case :—

Patient is a strong, very muscular man, æt. 60 ; lost left eye about 45 years ago by a blow from a large piece of coal while working in a mine. The injured organ continued " sore " for a couple of months, when the irritation disappeared. The eye gave no further trouble up

to January last. About the end of that month, patient first noticed a "weakness" when he awoke in the morning; could not easily open the eye, and dreaded the light—symptoms which disappeared after applying cold water for 10 to 15 minutes, leaving, however, a feeling of tenderness to the touch.

The right eye first showed signs of irritation about five weeks later (beginning of March), looked red, was painful and intolerant of light. Treatment in the country being of no avail, and sight failing fast during the last 12 days, patient came to New York and presented himself at the Eye Infirmary in the following condition:—

Left eye, irregular, somewhat shrunken; its cornea opaque, painful to the touch of a probe, which meets with a very hard mass, about $1\frac{1}{2}$ *mm.* broad, corresponding to the ciliary ring; moderate conjunctival and subconjunctival injection inwards and downwards.

Diagnosis: Fresh choroiditis or cyclitis in an eye suffering from the results of old choroiditis under the form of either ossification or calcareous degeneration.

Right eye, deeply injected, very painful, exceedingly sensitive to light; cornea and aqueous humor hazy; pupil contracted by numerous fine posterior synechiæ; vitreous cloudy; only large vessels of fundus visible; he counts fingers with difficulty at a distance of from three to four feet.

Diagnosis: Irido-choroiditis, very likely sympathetic.

The stump of left eye was at once removed, discovering the pathological processes which will be described hereafter; the right eye improved rapidly under atropine, darkness, mild aperients, and good diet. Patient insisted on leaving the city on the 17th day after enucleation, in spite of our remonstrances. Cornea clear, pupil dilated ad maximum; traces of former adhesions plainly visible as black spots on anterior capsule; vitreous transparent; retina still veiled, its vessels, however, can be followed without much difficulty almost to the periphery; papilla red but well defined; vision $= \frac{4}{15}$; field of vision shows slight concentric limitations.

Examination of the enucleated left eye-ball.—The specimen, hardened in Müller's fluid, revealed no abnormality in its outer coat; the diameter of the cornea, for instance, was 12 mm., as usual. It had been opened by an equatorial section. The posterior half consisted exclusively of the sclerotic, which was entirely denuded of the inner membranes, and showed only in its centre the intraöcular end of the optic nerve, with a small portion of soft and folded retina, the layers of which were still traceable, and, in particular, the nervous fibres surprisingly well preserved. The *choroid* had been taken away, no ossification or other abnormality *having been noticed in it.* A meridional section exhibited the condition represented in Figure 1, Tab. I. A hard septum (*a b*) stretched transversely through the anterior half of the globe. It arose from the anterior choroidal and posterior ciliary regions, reaching on one side (*a*) to the commencement of the ciliary processes, on the other (*b*) only to the middle of the pars plana corporis ciliaris. Its insertion was very unequal in breadth, varying between one and five millimetres. The septum, being thickest at the periphery, became gradually thinner towards the ocular axis (*c*, Fig. 1), where, in fact, it consisted only of the posterior lens capsule united with the hyaloid membrane. Its peripherical parts were granular, grating under the knife (osteoid tissue, *a*, Figs. 1 & 3), and were enveloped by a thin fibrous layer (*d*, Figs. 1 & 3). The crystalline lens (*o*, Fig. 1), being shrunken and applied to the central portion of the septum, was removed to

some distance from the iris (*i*, Fig. 1). In this manner a free space, filled with fluid, was formed between the septum and lens posteriorly, the ciliary processes (*p*, Fig. 1) laterally, and the iris anteriorly.

The anterior parts of the ciliary processes and the iris were apparently free from changes. The ciliary body could be lifted up from the sclerotic, thus exhibiting the ciliary muscle (*m*, Fig. 1), which seemed to be changed. In addition to the remnant of the retina at the optic nerve entrance before mentioned, only the anterior portions of this membrane were preserved, being in connection with the posterior edge of the septum (*r*, Fig. 1. In this drawing the whole retina is represented detached from the choroid). The microscopic examination, therefore, showed a condition not unfamiliar to ophthalmologists, namely, a plastic exudation springing from the ciliary region, and extending transversely through the vitreous body immediately behind the crystalline. It could be inferred, from its hardness, that either calcification or ossification had taken place in it. The origin of the exudation being in the region of the ora serrata and not at the ciliary processes, the retraction of the crystalline, and the integrity of the iris, are very peculiar and important features of this case.

The *microscopic examination* confirmed the presence of ossification, and revealed the following particulars worthy of notice, concerning the structure of the inner coats of this eye. In the *retina* there was a great increase of connective tissue fibres, which were slender and of a reti-

culated arrangement. The inner and outer granular layers were blended, the granules themselves diminished in number, but not in shape. The outer limiting and columnar layers were replaced by finely fibrillous undulating bundles of connective tissue. These changes are peculiar to what is termed *interstitial retinitis*, and were present both in the inner and outer layers.

The portions of the *choroid* behind the septum, which had remained with the specimen, showed no material change, except well-marked conditions of exudative inflammation in the vicinity of the septum. The capillary layer (*c*, Fig. 2, Tab. I.) was considerably thickened, and transformed into connective tissue, with larger and smaller vessels of irregular arrangement (*v*, Fig. 2). The hyaline membrane of the choroid (*h*, Fig. 2) was puckered, and perforated at different points by the proliferating connective tissue of the inflamed choroid, which extended as a thick false membrane (*f*, f_1, f_2, Fig. 2) into the vitreous, constituting the transverse septum before described. The choroidal pigment layer (*e*, Fig. 2) was broken up and irregularly dispersed through and upon the fibrous exudation. This exudation showed various stages of development. Near the hyaline membrane it (*f*) was of the same structure as the altered chorio-capillaris. A fibrillous matrix was densely filled with lymphoid bodies—small round cells with distinct large nuclei —and, in a less quantity, with spindle-shaped and stellate cells. Oval spaces filled with rows of lymphoid cells were numerous in this newly organized exudation, and

blood-vessels (*b*) of various size pervaded it freely. Farther from the choroid the structure of the septum was different. Its outer layer (f_1, Fig. 2, *f* and *d*, Fig. 3) consisted of ordinary soft connective tissue, having a ground-work of delicate fibres in which numerous small round and fusiform cells were embedded. The fibres were tough in the centre (f_2, Fig 2), and arranged in undulating bundles, with a smaller amount of cells and blood-vessels. The different stages of *ossification* could be followed in this central portion with remarkable clearness. In the places where the septum was thinnest, such as is represented in Fig. 2, there lay, in the middle of the fibrous tissue, a well-defined mass of denser structure (*m*, Fig. 2), which was less transparent, and less striated than the surrounding tissue. It appeared, under low magnifying power, to be homogeneous and finely granular; under high power it dissolved into a delicate fibrillated and minutely dotted matrix, in which small round and elongated cells were situated at regular intervals. This was the first stage of the ossific process. It could be easily followed up into the second, which is represented in Fig. 3, Tab. I., and has been so admirably described by R. Virchow under the name of *osteoid tissue*. The dotted appearance—chalky infiltration—of the matrix was better defined; the fibres had disappeared, and at regular intervals oval bodies, with short offsets and homogeneous contents, but without any nuclei, were visible. These bodies were connective tissue corpuscles in process of transformation into bone corpuscles. *Me-*

dullary spaces (*h*, Fig. 3) were so numerous and large in most places that the osteoid tissue enclosing them was more like a stiff trabecular network than a solid compact mass. The medullary spaces were filled with delicate connective tissue, enclosing many small round cells, and were pervaded by numerous blood-vessels (*v*, Fig. 3). Some of the medullary canals were partly, and others entirely, filled with large fat-cells. At some points on the border of the osteoid tissue small clusters of calcifying cells (*m*, Fig. 3) were seen isolated in the ordinary connective tissue; but in most places the infiltration with chalk was indicated by a well-defined, though irregular outline (*o*, Fig. 3). At very few points only of this specimen I met with *fully developed osseous substance*, showing the characteristic ragged anastomosing bone corpuscles.

Near the ocular axis the ossification ceased. Fibrillous connective tissue, continuous with the ossific substance, formed a false membrane (*f*, Fig. 3) behind the posterior lens capsule (*c*, Fig. 3). It became thinner the more it approached the centre of the posterior capsule, which was completely free from any deposit The commencement of the false membrane exhibited a sparse network of fusiform and stellate cells with large oval nuclei, a small body, and exceedingly long offsets. Some of them lay parallel, others intersected one another under various acute angles. Newly formed blood-vessels and lymphoid bodies were nowhere wanting, not even at the limits of the new formation of connective tissue corpuscles.

The crystalline lens, iris, ciliary muscle, cornea, and sclerotic manifested no structural alterations. The supra-choroid was also normal, with the exception of that portion lying near the septum, which participated, together with the outer choroidal layer, in the inflammatory process. The nerves of the ciliary region were very abundant and unchanged, so that neither from their number nor their caliber any degree of atrophy could be suspected. I should mention further, in opposition to Stellwag (Treatise, p. 232), that the nervous fibres had retained their normal medullary sheath.

I am likewise indebted to Dr. H. Althof for the examination of *two other eyes* affected with ossification from the choroid. Both were remarkable for the thickness of their bony shells, and the simultaneous occurrence of calcification and ossification. In both cases the detached retina penetrated, like a cord, through a round and smooth aperture in the ossific deposit, which, at this point, was 5 mm. thick. Exudations from the iris had undergone chalky degeneration. These specimens, having already gone through different hands, were unfortunately no longer complete enough to determine the origin and extent of the morbid products they contained.

The two following specimens I owe to the kindness of my friend Dr. G. Reuling, surgeon to the Maryland Eye and Ear Infirmary at Baltimore.

The first was the eye of a man, 45 years of age. It had been injured, five years previously, by a piece of metal, on the occasion of the explosion of a gun. The iris and lens had been wounded. Detach-

ment of the retina, and irido-cyclitis of the injured eye, and sympathetic affection of the other, followed, on account of which the former was taken out by the surgeon, who sent it to me, well preserved in Müller's fluid.

The eye-ball was somewhat smaller than natural, the cornea reduced to about half its normal diameter. The globe, opened by an antero-posterior section, displayed the following conditions (See Fig. 4, Tab. 1). The iris (*ir*) and anterior surface of the shrunken and degenerated lens (*l*) lay on the posterior surface of the cornea. The ciliary body, especially the ciliary muscle (*c*), were easily discernible. The retina was detached in the shape of a funnel, with its apex at the optic disc and its anterior insertion at the posterior end of the ciliary body, as usual. The retina enclosed a soft, granular-looking tissue (*v*), the anterior boundary of which was indicated by a curved line (*m*) stretching transversely through the globe. The centre of this line felt hard when touched with a needle. It was intimately connected with the remnants of the crystalline (*l*), which felt dense but not calcareous. Between the shrunken lens and the ciliary body there was a space (*n*) filled partly with fluid, partly with loose connective tissue. The choroid was adherent to the sclerotic; it seemed somewhat atrophic, had, however, no deficiencies of pigment; but very minute granules projected over its surface. In its posterior part, which was perforated by the coarcted retina (*r*), there lay an irregularly shaped hard thin plate (*p*), 11 *mm.* in length and 6 *mm.* in

breadth. It was evidently situated in the choroid, for its peripheral portion was completely and regularly covered with choroidal pigment, while the central portion had a smooth whitish, finely striated surface which was slightly raised over the peripheral portion. An isolated small hard plate was found more anteriorly in the choroid, and still another one forming a partial ring at the periphery of the transverse septum, with which the displaced retina was connected.

The microscopic examination showed that this ring consisted of osseous tissue. Its relations to the surrounding coats of the eye were identical with those found in the second case, to which Figs. 1 to 3 of Tab. I refer. The most anterior part of the choroid was thickened by connective tissue which originating in its capillary layer, perforated the epithelium (as in Fig. 2), and extended as a false membrane inward; differing, however, from Case 2 by not stretching horizontally through the globe, as in Fig. 1, but by being drawn forward. In some places its peripheral parts were nearly in contact with the ciliary processes. Ossification in this false membrane was not complete. In about one-half of the peripheric part well-developed bone was found; in the other half, only connective tissue, with small plates of osteoid substance. A small osseous tumor was observed in the centre of the transverse false membrane, lying behind the iris and the disorganized and shrunken lens. Choroidal pigment had been carried to this place by the stream of exudation, if the extension of the newly-

formed cellular elements may be called so. The lens showed remnants of fibres distorted in the most irregular manner, fat, and misshapen cellular remnants. Its capsule was wrinkled, but unaltered in structure. The stroma of the iris was mostly transformed into connective tissue. The pigment layer, however, was remarkably well preserved. A dense fibrous mass was found behind it, closely connected with the remnants of the lens. *The ciliary body*—muscle and processes—was, in most places, in an entirely healthy condition. It did not participate in the process of proliferation and ossification which arose from the anterior part of the choroid, and by being drawn forward came almost in contact with the outer surface of the ciliary processes. Had the disease been allowed to continue its march this contact would have been effected, and the outer surface of the osseous plate would have shown a cast of the ciliary processes, as Arnold Pagenstecher has observed in one specimen. At one point only the ciliary body was degenerated, participating with the iris in the formation of cicatricial tissue. This was the point at which the foreign body had injured the eye, and from which the sympathetic irritation must have started.

The choroid was not materially changed in its middle and anterior parts, with the exception of its anterior edge which has been already described. The finely granular appearance of its inner surface before mentioned resulted from extensive *colloid* deposits on its hyaline layer. These colloid bodies differed greatly in size and form:

some were no larger than the pigmented epithelial cells of the choroid, others were 0.5 *mm.* in diameter. They were mostly globular, but in some instances much elongated, here and there projecting like vermiform processes over the surface of the choroid. The smaller ones were evenly covered with epithelium, the larger had a smooth unpigmented surface. They were also seen on that part of the choroid (*p*, Fig. 4, and *c*, Fig. 5) which contained the osseous plate. This plate (*o*, Fig. 5) was situated in the capillary layer (*n*, Fig. 5) of the choroid, all the other layers of this membrane being well preserved. The capillary layer itself was enlarged by intussusception of new-formed cells, fibres, and blood-vessels. The nutritive canals (*m*, Fig. 5) of the bony plate extended from the outer layers of the choroid to the inner border of the chorio-capillaris. Small vessels arising from the outer choroidal vessels (*v*, Fig. 5) penetrated into these nutritive canals, being enveloped by delicate connective tissue, like the vessels ramifying between the nerve-fibre bundles of the opticus. The osseous corpuscles in the bony plate were well developed, but very delicate. They had an infinite number of offsets by which they anastomosed with one another. Immediately after the preparation had been taken out of the alcohol and placed under the microscope, the corpuscles and their offsets, in some places, appeared as dark as if they had been taken from an old piece of bone; but soon after glycerine had been added they became transparent, and showed, in their interior, very distinct nuclei or cells, the outlines of which were at some dis-

tance from the walls of the lacunæ. When the preparations were mounted in some other preservative fluids—for instance, Canada balsam—the bone corpuscles became black as usual. The conversion of the fibrous tissue into osteoid and osseous substance was immediate; the chalky incrustation, in this as in most specimens I have examined, was, however, so meagre that sections could be easily made with the knife.

The *remarkable features of this case, summed up briefly, are the following:* A foreign body had wounded the cornea, iris, lens, and ciliary body. The lens had become partially absorbed and disorganized. Plastic chronic inflammation in the iris and ciliary body had been the result, causing, after the lapse of five years, sympathetic inflammation in the other eye. The choroid had undergone osseous degeneration in its capillary layer to a considerable extent around the optic nerve entrance. From its anterior edge arose a false membrane which extended across the eye-ball, being, however, drawn forward by the shrinking cicatricial tissue in the anterior part of the eye. This membrane was ossifying in its peripheral and central parts. The ciliary body was healthy, except in that part which had been injured by the foreign body. The hyaline layer of the choroid was studded with colloid globules. The retina was detached and coarcted in the shape of a funnel, attached anteriorly to the transverse false membrane, posteriorly to the optic nerve. It was degenerated by a great increase of its connective tissue elements, both fibres and granules. The

remnants of the vitreous body enclosed by the retina and the transverse septum were completely metamorphosed into connective-tissue fibres and cells.

The second of Dr. Reuling's cases was that of a man 38 years of age; his eye-disease had likewise a traumatic origin. No other particulars of his history were given.

The specimen (Fig. 6, in the text) exhibited conditions very similar to those of the preceding case. The ossification was still more advanced. The retina was likewise detached and compressed into a solid funnel-shaped mass (*r*, Fig. 6), arising with its small end from the optic disc, perforating the bony shell (*b*) of the choroid, passing through a serous fluid (*s*) which filled the posterior space of the eyeball, and inserting itself with its broad end into a hard transverse septum (*h*) behind the lens (*l*). The osseous plate (*b*) of the choroid was thickest—1.5 *mm.*—around the optic disc. Its extension was very unequal on both sides, reaching on the one only as far as 3 *mm.* from the disc, on the other over the whole length of the choroid, and uniting with the transverse septum, which arose from the anterior margin of the choroid and stretched itself across the globe. This septum was thickest in its centre, where it corresponded to the remnants of the vitreous body enclosed by the retina. It contained in its middle a black

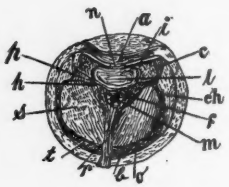


Fig. 6.

irregular *foreign body*, a piece of iron (*m*), which was encapsuled by white fibrous tissue (*f*). Its peripheric portion (*p*) felt hard, bony, in its whole circumference. In front of the septum lay the crystalline lens (*l*), its size but little diminished, and its consistence unaltered. The ciliary body (*c*) was situate at the side of the crystalline lens, and likewise before the transverse septum. It was as soft as usual, and had on the whole a normal appearance. The ciliary body and the adjacent part of the choroid (*ch*) were separated from the sclerotic, as if they had been drawn inward by the contraction of the transverse septum. The cornea (*a*) was depressed and in immediate contact with the iris, which, to the naked eye, did not appear abnormal. The pupil was occluded by a thin, transparent false membrane (*n*).

Microscopic examination revealed the presence of osseous tissue in all the parts which felt hard to the exploring needle. The ossification originated in the capillary layer of the choroid, and attained its greatest thickness in the neighborhood of the optic nerve entrance (*b*, Fig. 6). The outer layers of the choroid (*o*) were little changed, but the inner layers,—hyaline and epithelial strata,—in the posterior parts, were replaced by dense fibrous connective tissue (*t*). The anterior edge of the choroid was again the seat of plastic inflammation, the products of which extended as a false membrane transversely through the globe, in the manner before described. Osteoid and osseous tissue was found

in the peripheral portions of the septum (*p*). The cornea, iris, ciliary body, and crystalline lens did not reveal any extraordinary conditions. An abundance of small cells and delicate fibres was present in the iris, indicating that this membrane was in a state of inflammation, of which the pupillary membrane was an additional symptom. I could not have traced the course which the foreign body had taken without totally sacrificing this very instructive specimen. It seemed that the foreign body had passed through the sclerotic, and had been immediately suspended and encapsuled in the vitreous body behind the lens. This is very unusual, as we know, chiefly by Dr. Berlin's researches, that heavy foreign bodies, such as pieces of stone and metal, when they have once entered the vitreous chamber, commonly strike the opposite wall, from which they are repelled and sink to the bottom of the globe. The sojourn of the foreign body in the eyeball, together with the presence of irido-choroiditis, suffices, apart from the extensive ossification, to account for the sympathetic ophthalmia in the other eye. With regard to the ossific process, this case shows the same general conditions as the preceding cases.

The following case came, not long ago, under my own observation, and served as an example to utilize, with regard to diagnosis and treatment, the experience and results derived from the study of the foregoing cases.

Mrs. P., of New York, nineteen years ago contracted in her left eye

an intense acute inflammation, which, judging from the symptoms she described, was an iritis. The inflammation subsided in the course of two weeks, but left tenderness and great impairment of vision behind. Every now and then this eye would inflame again in a less degree, and cause irritability of its fellow. Twelve years ago both eyes suffered simultaneously from a severer attack, from which the right recovered, but the left became blind. Off and on during the last twelve years, the left would be tender and slightly bloodshot, the right sympathizing, dreading the light, weeping, and being easily fatigued. Its sight, however, continued good, and she could easily read and sew with it, until the commencement of September, 1870, when it became severely inflamed, painful, especially during the night, and very much impaired in sight. A fortnight after she came to the New York Ophthalmic Institute for relief, her right eye exhibiting the symptoms of severe iridochoroiditis. There was marked circum-corneal injection; not uniform however, the outer part being much whiter. The iris was dirty gray, its pupillary edge completely adherent. The pupil dilated somewhat by the use of atropine, which stretched the circular russet synechiæ without tearing any. The lens appeared diffusely grayish, but showed no cataractous dots or stripes. The interior of the eye could be dimly illuminated, but none of its details were recognizable. $S = \frac{2}{200}$, F complete, and Tn. The other eye was only slightly injected, and but little tender on pressure. As mercurials, leeches, aperients, and atropine were used for a week without any benefit, and the pain in the right eye became so great as to deprive her of sleep during the whole night, the removal of the left eye was decided upon, in the belief that the affection of the right had its origin in its sympathy with the left.

Immediately before the enucleation, the left eye was in the following condition. An opaque stripe was in the lower half, and apparently on the posterior surface of the cornea. The iris was greenish-yellow, bulging in its lower part, and bearing on its anterior surface two small velvety excrescences. The pupil was small, and filled with brown pig-

ment. The size and shape of the globe were unchanged; the episcleral vessels tortuous and numerous. The posterior region of the globe felt hard like wood to the touch, the hardness terminating in an irregular line, 4 to 5 *mm.* behind, and parallel to the corneal margin. The globe in front of this line was of normal tension. Pressure upon the ciliary region was painful; not so, however, upon the hardened portion behind it.

The globe was extirpated on the 23d of September. The wound healed well. The sub-conjunctival injection and tenderness of the right eye greatly diminished for a few days, and vision improved. Afterward the patient felt severe pain in the eye nearly every night, lasting for one or several hours, while the days were pretty comfortable. The anterior chamber continued shallow, and the iris discolored. The pupil dilated only moderately by the use of atropine. The fundus could be illuminated, but its details were not recognized. Vision was doubly or three times as good as before the operation. She was treated with atropine, leeches, hydrate of chloral—opiates were not tolerated—and iron with rhubarb as a tonic. On the 8th of November she had a relapse. The anterior chamber was turbid, the iris discolored, vision reduced from $\frac{1}{20}$ to $\frac{1}{100}$; great pain in the eye and head. This relapse yielded to an application of leeches, and at present, November 21st, the eye bids fair to recover, with S = $\frac{1}{20}$, the refracting media not yet having cleared up.

The eye-ball was placed in Müller's fluid, and, some days after, in absolute alcohol. An antero-posterior section exhibited the following conditions (Fig. 7 in the text). The whole inner surface of the choroid was invested with a bony shell (*b*), of a thickness varying from one-half to five millimetres. This shell ceased immediately behind the ciliary processes (*c*), and continued into a transverse septum lying behind the lens. At the

periphery of the septum there were some hardened portions. The retina (*r*) was detached, united anteriorly to the peripheral part of the septum, posteriorly to the optic nerve, after having pierced the thickest portion of the bony capsule. The detached retina was not so completely coarcted as in the former cases, but had some lateral expansion, admitting of the folds being separated from one another. There was, however, no free space between the folds, these lying in immediate contact with each other, so that the vitreous body had been entirely absorbed. The hard shell was covered with a delicate white fibrous coat, between which and the retina there was a watery fluid containing coagulated albumen. The ciliary body was thickened, the inner surface of the ciliary processes irregular, and here and there coated with soft fibrous exudation (*f*). The iris was likewise thickened. The whole substance of the ciliary body and iris was soft, no hardened concretions being felt with the needle. The lens (*l*) was reduced in diameter, but not in thickness. The anterior corticalis contained a thin, and the posterior corticalis a thick layer of chalky deposit. The lens capsule, however, was normal throughout. Between the lens and ciliary processes there was a space of two to four millimetres (*f*), filled

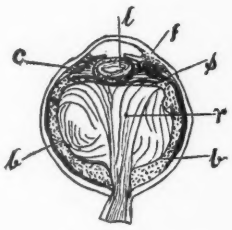


FIG. 7.

partly with fluid, partly with soft fibrous exudations. *The microscopic examination* of the specimen did not reveal any new facts with regard to the formation of bony substance, but furnished an additional example of the constancy of the conditions under which we saw ossification take place in the preceding cases. It was another example of an eye suffering from old irido-choroiditis. The ossification had its seat in the inner layer of the choroid, the outer choroidal layers showing symptoms of chronic inflammation, viz., disarrangement and atrophy of their natural elementary parts, together with hypertrophy of connective tissue. The bony shell was of the usual structure. It terminated at the ora serrata, from which region here also a transverse false membrane arose in which ossification had begun at the periphery. The retina was disorganized, connective-tissue fibres and cells having replaced its natural elements to such a degree that the different retinal layers were nowhere recognizable. The lens being shrunk laterally, but not diminished from pole to pole, is evidence that its suspensory ligament had lost its physiological action to flatten the crystalline body. The centre of the lens was normal; its periphery had undergone chalky degeneration without the not infrequent interposition of connective tissue between the capsule and the chalky deposit. The changes in the ciliary body and iris were important, as they must have been the cause of the sympathetic inflammation of the other eye. They consisted in an abundant infiltration of the tissue of the ciliary muscle,

the ciliary processes, and iris, with lymphoid bodies—formative cells—in addition to which newly formed connective tissue was observed, replacing a part of the original stroma, proceeding through the epithelium of the ciliary processes into the posterior chamber, and sprouting from the anterior surface of the iris to form the velvety excrescences seen upon the iris before the eye was extirpated.

Here we see, more than in any of the other cases, a well-pronounced inflammation of the ciliary body and iris to account for the origin of the sympathetic inflammation in the fellow-eye. The sympathetic inflammation was also of a higher development than in any of the preceding cases. How long the inflammation had existed in the eye primarily affected until it waked up such a severe inflammation in the other, cannot be determined. The repeated attacks of temporary inflammation in the first eye had often been accompanied by slighter irritations in the second, which, however, had passed without causing notable injury, until lately the affection in the second eye had reached such an intensity as to threaten its preservation, and necessitate the removal of the first.

If I review, with regard to the questions set forth at the beginning of this paper, these cases, and avail myself of the literature as far as it is accessible to me, I arrive at several conclusions of some general value.

Nowhere has there been found true ossification in the crystalline lens, retina, cornea, iris, and vitreous body. The osseous formations which have been described in the

vitreous must be considered as metamorphoses of plastic exudations, false membranes, originating from some part of the uveal tract. *Calcareous deposits*, on the other hand, *have been observed in all the structures of the globe.*

*True ossification has only been found in exudations from the choroid.**

When the process is still in a low stage of development, the ossification takes place in small plates, which lie in the chorio-capillaris, and are covered by the hyaline membrane and the epithelium of the choroid, *as is illustrated in* Fig. 5, Tab. I., and by Arnold *Pagenstecher* in Arch. f. Ophth. VII., 1, Plate II., Fig. 5. If, however, the morbid action be further advanced, then the more abundant exudation wrinkles and perforates the hyaline membrane, invests the inner surface of the choroid, and is gradually and slowly converted into osteoid tissue. The retina remains sometimes in immediate contact with the inner surface of the osseous capsule (Wardrop in Mackenzie's Treatise, IV. Ed., p. 677), but it is generally separated from it, retaining, however, its attachments to the ora serrata and the optic disc, perforating the bone shell in the latter place. The abrupt termination of the osseous capsule at the ora serrata made me think that the chorio-capillaris plays an essential part in the pro-

* The best and, as far as I know, also the most recent description, together with a *full account of the literature* on ossification in the eye, is given by Dr. Arnold Pagenstecher, in a paper prepared in the laboratory, and under the auspices of Professor Heinrich Müller, in Würzburg, published, in 1860, in *Graefe's Archives*, Vol. vii., part 1, pp. 99–118. Since this work is easily accessible to everybody, I refrain from quotations of literary sources.

duction of the ossific deposits. Ossification, I argued, being an active process, and requiring much nutritive material, is dependent on the chief source of nutrition of the interior of the globe, namely, the capillary layer of the choroid, which, according to Dr. Theodore Leber's investigations, terminates at the pars plana corporis ciliaris, that is, in the region of the ora serrata. These views have been confirmed by further inquiries.

The continuation of the ossifying connective-tissue coat covering the inner surface of the bone capsule into a false membrane, stretching like a septum from the ora serrata transversely through the vitreous body, serves to bring the occurrence of osseous layers in this region into connection with the chorio-capillaris. Henry Müller and Arnold Pagenstecher (l. c., p. 102) also describe the occurrence of such a septum at the same place, and in connection with a bony shell terminating at the ora serrata. The second case described in this paper shows (Figs. 1 and 2, Tab. I.) the primary plastic exudation originating in the anterior part of the chorio-capillaris, perforating the hyaline membrane and pigmentary layer, and expanding into the vitreous (Fig. 2). It was distinctly visible that the ciliary body was not the starting-point of the exudation, as it lay before it and was not materially changed.

This false transverse membrane, however, although starting from the terminating portions of the chorio-capillaris, increases in thickness as it grows, and ultimately reaches the ciliary processes, the zonula Zinnii, and crys-

talline lens, which by the contraction of the newly-formed tissue may be drawn backward. Arnold Pagenstecher observed in one of his cases that the anterior portion of the bony shell had on its outer surface distinct impressions from the corona ciliaris. This also proves that the bone production did not originate in the ciliary processes, but was only in contact with them during its solidification. In our fifth case the ossific septum was drawn forward toward the cornea, thus coming in contact with the ciliary processes which were found unaffected. In our other cases the osseous deposit did not reach the ciliary processes at all, but left them intact in their position, although it drew the lens backward (Figs. 1, 6, and 7). It is extraordinary that in one of our specimens—the second described—the ossification was not noticed on the choroid proper, but was confined to the false membrane behind the crystalline. If the cursory examination made before the choroid was taken off is to be considered sufficiently reliable for admitting that no osseous plates were present in the choroid, the process of plastic exudation, of which ossification is only the consequence and final stage, must have been limited to the anterior portion of the choroid. This in reality seems to have been the case, as the exudation—represented in Fig. 2—was circumscribed, and the small remnants of choroid which I found behind it manifested no material change in structure.

To complete the description, I should refer here to a most interesting specimen described and illustrated by a

woodcut in Stellwag's Treatise (Fig. 35, p. 233). A thick osseous shell invested the choroid from the optic disc to the commencement of the ciliary body; thence turned its course inward behind the crystalline, forming thus a complete shell of a triangular shape, with about an equal thickness throughout. The ciliary body here, too, lay before the shell, and did not participate in the ossific process. The case represents the last stage of ossification in the eye, as far as I could follow up the reliable literature. The three latter of our cases (Figs. 4, 6, and 7) bear the same features as Stellwag's case, with this difference only, that the osseous shell was not so complete nor so thick as in his. In most earlier writers petrification and ossification are not distinguished from each other; and as both may occur simultaneously in the same specimen, as, for instance, in the third and fourth here reported, the deposits from the iris and in the lens described by the earlier writers must be taken for what they have been more recently found to be, namely, calcareous degeneration, until further evidence of ossification in these parts is furnished by the study of new cases.

Although the subject under consideration has not yet been sufficiently investigated, and the material at my disposal for original inquiry is too limited to yield results which might not be essentially modified by subsequent observations, *I arrive*, nevertheless, *in summing up* the facts contained in this paper, at the *following* not unimportant *conclusions*:—

Formation of bone in the eye is the consequence and final stage of *a plastic inflammation of the capillary layer of the choroid. It invests the inner surface of the choroid, and continues, in the form of a transverse septum*, through the anterior part of the vitreous humor, behind the ciliary body and the crystalline lens, without, however, involving these structures.

The *diseases which lead to ossific productions* are chronic inflammations of the interior coats of the eye, called internal ophthalmia by the earlier, irido-choroiditis by modern writers. It should be particularly mentioned that chronic plastic inflammation limited to the ciliary body and iris, irido-cyclitis, which is not uncommon, and leads to the formation of false membranes before and behind the crystalline lens, has not been found to generate osseous tissue. Nor has ossification been demonstrated as a sequel of iritis alone.

The *diagnosis* of ossification may be based upon the unyielding and hard condition of the posterior part of the globe, terminating about two lines behind the cornea, while the anterior region is soft, or at least impressible.

Simultaneous occurrence of *calcification* and *ossification* is frequent. Calcification may be diagnosticated by atrophy or degeneration of the iris, in combination with a shrunken white or yellowish cataractous lens.

As ossification does not involve the outer choroidal layers, nor the ciliary muscle and iris, it in itself is *not* to be dreaded as a cause of *sympathetic ophthalmia.*

The latter can only result from irido-cyclitis supervening as a complication to ossific choroiditis.

As long as an eye in which ossification is diagnosticated remains free from irritation, and its fellow also, the *removal* of the former, for fear of sympathetic inflammation, is *not indicated.* To corroborate this, I may state that I have not infrequently observed cases in which eyes bearing the signs of ossification, and even being tender to the touch on the ciliary region, were toler ated without annoyance for twenty, thirty, and forty years.

A CASE OF GLIOMA RETINÆ, OPERATED ON AT AN EARLY STAGE, AND EXHIBITING SOME NEW AND PECULIAR ANATOMICAL CONDITIONS.

By H. KNAPP.

(Plates II. and III.)

Intra-ocular tumors, especially glioma of the retina, have of late years excited the particular interest both of ophthalmologists and pathologists. All investigators, Virchow at their head, express themselves in such a way that further observations are still needed, and, above all, such as treat on the early stages of this important disease. The following case, therefore, may be found worthy of attention in more than one respect:—

Herman Pillat, of New York, a healthy-looking boy, 8 years of age, noticed, in October, 1868, on a particular occasion, that he could not see with his left eye. This occasion was that he had hurt his left eye with the corner of a book. His eye ached for about a quarter of an hour, then all symptoms of irritation disappeared, and he did not tell his parents of the injury until a week later they discovered, by accidentally closing his right eye, that the left was totally blind.

The appearance of the eye did not show any abnormality, the pupil, especially, being regular and movable. After about six months, however, they noticed that the pupil had become larger than that of the other eye, and presented a deep-seated reddish-yellow lustre when turned towards the light. At that time they consulted, as they told

me after I had enucleated the eye, H. D. Noyes of this city, who intimated to them that a malignant growth was developing in the eye, and the removal of the latter would soon become necessary. Alarmed by this statement, and hearing opinions more consolatory to them, the eye being, moreover, free from all redness and pain, they omitted to see Dr. Noyes again.

Early in October, 1869, the eye began to become now and then painful. About the middle of the month, while on a short sojourn up the Hudson, during bad weather, he caught a severe cold in his head and lungs. At the same time the eye began to become red, more painful, and watering. A week after, on the 26th of October, 1869, the patient was brought to me and showed the following condition:— General health, aside from the remnants of the cold, excellent. Right eye in every respect normal, only a little intolerant of light, and easily weeping. *Left eye* considerable subconjunctival injection; cornea very slightly hazy, and less sensitive than in the other eye; pupil immovable, dilated in the form of a vertical ellipse; iris of a dirty grayish-brown discoloration; globe hard (T_1); no perception of light. The pupillary field appeared dull and smoky. A dirty yellowish-brown reflex, particularly marked in the lower half, shone through the pupil, originating from a mass which, on examination with focal light, appeared free from blood-vessels, uniformly dull reddish-yellow, and lying immediately behind the transparent and a little advanced crystalline lens.

I informed the parents that a malignant tumor had developed in the eye, having just entered the stage when it leaves the organ and infects the system. The eye, I added, should be removed without delay, if they wished to save the life of their child. This firm declaration, being in conformity with what Dr. Noyes had already pointed out to them six months previously, made them immediately consent to the enucleation of the eyeball, which I performed the next day. No accident occurred. The wound healed without suppuration, and up to this time, December, 1870, the boy has shown no symptoms of a recurrence in the orbit or in remote organs, and has enjoyed excellent health.

Examination of the Enucleated Eyeball.

The globe was opened immediately after its removal, by an antero-posterior section beginning in the centre of the cornea. Although the knife was sharp, the lens was separated from the ciliary body, being united with the base of a funnel-shaped detached retina (*r*, Fig. 1, Tab. II. In this figure the parts are drawn according to their proper situation before the section was made). The anterior portion (*t*) of the retinal funnel represented a yellowish swelling reaching to the posterior capsule of the lens. The surface of the intumescence was wrinkled in some places, and slightly nodular in others, but *smooth* throughout. The posterior portion of the detached retina was folded, and studded with a few small, yellowish, hemispherical granules (t_1), the outer surface of which being likewise smooth. The choroid, ciliary body, and iris were in their proper positions, and exhibited no changes noticeable to the naked eye.

The globe was immediately placed in Müller's fluid, and, some months later, examined with the microscope. The lens and degenerated retina were now so well hardened that I could make an antero-posterior section, as represented in Fig. 1. There was a yellowish, soft, granulated tumor (*t*) immediately behind the healthy lens, measuring 10 *mm.* in its longest diameter. On the one side (*a*) it was blended with the membrane enveloping it; on the other its boundary (*b*) was separated from the retina by a space (*c*) filled with fluid (vitreous body

during life). In this space the inner surface of the retina, near the tumor, was slightly uneven, showing some papulose elevations or rather thickenings. The tumor itself could be raised from the retina for a certain extent, but further on was organically connected with it. The space (*d*) situated behind the tumor, and enclosed between the retinal folds, contained fluid (vitreous).

On *microscopic examination* the tumor (*t*) manifested itself as a pure glioma of the ordinary character:—small round cells, homogeneous scanty matrix, and many thin-walled blood-vessels. This was the invariable structure from the posterior boundary of the pseudoplasm to the layers adjoining the crystalline (*c*, Fig. 5, Tab. II., *gl. m.* Fig. 4, Tab. III.).

The posterior portion of the retina, which macroscopically exhibited no degeneration except the few small nodules mentioned before, was carefully examined. In fact, I used this whole portion of the specimen for making microscopic preparations in order to discover the starting-points of the new growth. At no point did I encounter entirely healthy retina. The inner layers of this membrane were in many places pretty well preserved. The molecular layer had disappeared. Both the granular layers were blended, so that no intergranular layer was visible. The internal and external limiting membranes were in good state of preservation. The rods and cones were missing, as is usual in detached retinæ. The radiating fibres were better preserved than any of the other constituent parts of the retina.

At many points in the detached retina I found all the nervous elements wanting. Both limitantes enclosed a frame-work of connective-tissue fibres, with its normal arrangement in the inner half, and an abundance of small round cells embedded in a scanty homogeneous matrix (*A*, Fig. 2, Tab. II.). The small cells were densely and uniformly accumulated in the outer half; they pervaded, however, the whole retina, being only more sparsely scattered through the matrix of the inner layers. In other places the retina was entirely metamorphosed into a homogeneous band, in which nothing but small cells, densely grouped together, were visible. All the changes described till now were not connected with any noticeable increase in the thickness of the retina, and represented what is called *diffuse gliomatous degeneration.*

The small semiglobular elevations (t_1, Fig. 1) on the outer surface of the retina were glioma tumors, arising from a wholly degenerated retina without affording an opportunity to detect their points of origin.

Not infrequently I found the detached retina swelled to about its double or threefold thickness, in the manner which is represented in Fig. 2, Tab. II. The nervous elements had disappeared, the radiating fibres were preserved, and the small cells crowded between them. Both limitantes well marked and pushed aside by the multiplication of the cellular elements. These consisted of granules absolutely identical in appearance to white blood-cells (*b*, Fig. 2). Some of them, showing a conspicuous nucleolus, bore the closest resemblance to the

nuclei of sarcoma-cells (*c*, Fig. 2). A small protoplasmic ring could be discerned in many of the cells. The blood-vessels (*a*, Fig. 2) were numerous, well filled with red blood corpuscles, while white blood-cells were crowded between the fibrous and muscular elements of the walls of the vessels (*d*, Fig. 2). A free space (*e*, Fig. 2), the so-called perivascular space or lymphatic sheath of the vessel, looked very much as if it had been produced by the contraction of the blood-vessel and the transudation of blood-plasma.

A number of homogeneous, translucent hyaline bodies (*f*, Fig. 2), of irregular, mostly oval form and dull oily lustre, were scattered through the mass of small cells. These bodies are of frequent occurrence in all forms of destructive retinitis, and commonly described as cells having undergone *colloid* metamorphosis, but they may be *myelin* coalesced after the destruction of the nervous elements.

Here and there choroidal pigment, in little clusters of granules or regular cells (*g*, Fig. 2), was seen scattered through the external layers of the retina. In some places of the anterior portion of the retina the choroidal pigment was so abundantly strown between the cells of the degenerated retina, as to indicate that *the morbid process had been active before the retina became detached.*

I studied the anterior portion of the detached retina with special care. Fig. 3, Tab. II. represents a transverse section through the periphery of the tumor immediately behind the lens (*pc*, *a* Fig. 1, Tab. II.). The

smooth boundary consisted of the anterior part of the retina (*rr*, Fig. 3) up to the ora serrata (*o s*, Fig. 3), and its prolongation the *pars ciliaris retinæ* (*pc*, Figs. 1 and 3). *The latter was detached from the ciliary processes*, blended with the zonula Zinnii, and inserted itself into the equator of the crystalloid. When examined with an immersion system it showed the normal band-like fibres quite unaltered, containing hardly any more granulated nuclei than in the normal state. The ora serrata (*o s*, Fig. 3), which shows a small thickening also in the healthy condition, was moderately filled with small round cells. The adjoining retina (*r r*, Fig. 3) consisted only of radiating fibres, abundantly interspersed with the same kind of cells. Both the limiting membranes were well preserved. This anterior portion of the retina and the pars ciliaris enclosed the elements of the pseudoplasm, either in their ordinary condition (*b* b_1, Fig. 3) or in fatty degeneration (*c c*, Fig. 3). Blood-vessels (*d*, Fig. 3) were numerous in the whole mass.

The retina, not far from the ora serrata, was the seat of conspicuous changes. It swelled by multiplication of small cells, *the origin of which*, in many places, could be *distinctly traced to the inner granular layer*. Fig. 3, at its end B, and Fig. 4, Tab. III., give faithful representations of this condition. B, Fig. 3, shows a circumscribed accumulation of glioma cells in the neighborhood of a blood-vessel, extending thence, in two unbroken rows, through the inner retinal layers beyond the limitans interna into the vitreous body (b_1, Fig. 3). The outer

granular layer was likewise invaded, but in a less degree, and without perforation of the outer limitans. I could here reproduce also such figures as Iwanoff publishes (Arch. für Ophth. XII., Taf. II., fig. 9), where the outer layers were but little changed, and in the inner layers there was a production of small round cells grouped around a blood-vessel, and pervading the membrana limitans interna. Also conditions like those in his figure 10, l. c., in which the outer layers were tolerably normal, at least not increased in thickness, whilst the inner layers were densely filled with lymphoid cells, were found in some of my preparations. Figure 3, Tab. II., has many points in common with them. In the vast majority of the preparations taken from the anterior portions of the retina where the tumor was located, the *elements of the pseudoplasm originated in the inner granular layer.* Both granular layers, indeed, were blended, and the intergranular layer had disappeared, but the primary clusters of the newly formed elements never occupied the outer layers. Fig. 4, Tab. III. will give a good illustration of this condition. The limitans interna (*li*) was mostly well preserved; the same can be said of the radiating fibres (*rf*), nervous fibres (*n*), and ganglionic cells (*g*). The remaining layers of the retina (*h*) were metamorphosed into a regular set of transverse (radiating) fibres, interspersed with granules. The limitans externa (*le*), in many places, was well marked, in others destroyed by the proliferating tissue which here and there spread from the outer granular layer (*ak*), but did not develop

into large masses. At various points there were distinct *inflammatory changes in the tissue of the retina*, namely, an increase of connective tissue, the fibres of which were broader, freely anastomosing with one another, and interspersed with many disconnected lymphoid bodies, thus notably augmenting the transverse section of the retina.

Entirely different from these inflammatory conditions was the appearance of the glioma clusters. In the middle, and towards the inner side of the more or less changed outer layers of the retina, lay dense accumulations of small round cells (*gl*, Fig. 4), pressing the adjoining tissue aside, extending into the outer granular layer (*ok*), perforating the outer limitans, and pouring in large, compact masses into the free vitreous space (*mm*). Blood-vessels were numerous, both in the retina where the glioma originated (*v*) and in the free tumor. All this leaves no doubt, *that the inner granular layer was the starting-point of the bulk of the pseudoplasm, although a limited growth* was also observed in the *outer granular layer* (*ak*, Fig. 4), and in the *inner* (molecular, ganglionic, and nervous) *layers* of the retina.

Another peculiarity of this tumor puzzled me very much at first, but proved, on closer examination, of extraordinary interest. Being particularly desirous to obtain good microscopic preparations from the borders of the tumor, where its outer surface was enveloped or rather formed by the retina, I found nearly always that *the outermost layers of the tumor*—its outer smooth sur-

face—*were unmistakably the inner layers of the retina.* Fig. 4, Tab. III., for instance, is taken from the surface of the tumor, at *b*, Fig. 1, Tab. II. The limitans interna (*li*, Fig. 4), the fibrous and ganglionic layers are turned towards the free space (*c*, Fig. 1) between the tumor (*t*) and the detached retina (*e*). The outer (granular) layers of the retina (*ak*, *ok*, Fig. 4), the bulk of the neoplasm (*m*, *m*, Fig. 4), were turned towards the optical axis, that is, grew inwards into the vitreous space. In search of the cause of this singular perversion I found that I was able to raise, with the needle, the tumor (*b*, *t*, Fig. 1) over a considerable extent from the surrounding retina (*e*, Fig. 1). At one point (*a*, Fig. 1), however, I was unable to do so; here I found the tumor no longer surrounded by the retina. In fact, the whole growth, with the exception of this last-named point, *was enveloped by the detached retina and its prolongation, the pars ciliaris* (*e*, Fig. 1). The outer surface of the tumor, wherever it was surrounded by the detached retina, consisted of the inner layers of the retina, so that the limitans interna, forming the surface of the pseudoplasm, was either in contact with the limitans interna of the enveloping retina, or separated from it by a free space filled with fluid, as seen in *c*, Fig. 1. This was evidently a remnant of the vitreous space, and communicated with the posterior remnants of the vitreous, which was confined by the posterior portion of the detached retina, *d*, Fig. 1.

There was, however, yet a third portion of vitreous

preserved, although materially altered in structure, namely, immediately behind the crystalline (*v*, Fig. 1). The posterior capsule (*b*, Fig. 5, Tab. II.), was unchanged in structure, as was also the whole crystalline lens. A layer of loose connective tissue (*aa*, Fig. 5) was interposed between the posterior capsule and the pseudoplasm (*c*, Fig. 5), the elementary parts of which indefinitely bordered on, and penetrated into, this newly-formed connective tissue.

The *choroid* was unaffected in its whole extent. Some detachment of the pigment epithelium in various places may partly be due to the morbid action of the gliomatous process in the retina, before its separation took place; the greater part, however, was certainly due to cadaveric maceration, and had, therefore, no significance. The *ciliary body* was normal, with the exception that the pars ciliaris retinæ, together with the zonula Zinnii, was torn from the surface of the otherwise normal ciliary processes. The *iris* was healthy throughout, and likewise the other tissues of the globe. I should make particular mention that sections through the *optic nerve outside the sclerotic* did not reveal the slightest trace of morbid changes.

According to the foregoing examination the *development* of this pseudoplasm must have been as follows. The origin of the foreign elements is found in small, more or less circumscribed clusters, lying in the inner granular layer (*gl*, Fig. 4, Tab. III.). These clusters were the most luxurious part of the new growth, and

extended in every direction, but principally towards and beyond the outer granular layer (*ok* and *m*, Fig. 4). At first the proliferating process advanced slowly, generating products of inflammation in the retina, viz., increase of its connective-tissue framework (*h*, Fig. 4), with a more or less complete destruction of its nervous contents (Fig. 2, Tab. II.). We may presume that in the early stage of the development of the new growth the retina remained adherent to the choroid, since Iwanoff has recently (Graefe's Arch. XV. 2, p. 69, etc.) described a case in which the retina, although degenerated to a greater extent than in our case, adhered everywhere to the choroid. The pseudoplasm, in Iwanoff's case, sprang from the *inner retinal layers*, and penetrated through the inner limiting membrane into the vitreous space. Such a condition had been described only once before (Manfredi). The main bulk of the tumor, as in our case, was located in the anterior portion of the retina. Iwanoff, adhering strictly to the opinion of Virchow, that glioma is a hyperplasia of the connective-tissue elements of the retina, believes that it may grow from all the retinal layers, since none of them is devoid of such connective tissue (neuroglia). Our case furnishes an additional example of the truth of this assertion. In many places I saw the outer granular layer being the seat of the proliferating elements (*ak*, Fig. 4), while the other retinal strata were exempt from them. At other points the inner layers of the retina were filled with glioma cells, which entered through the

limitans interna into the vitreous just as Iwanoff has described and illustrated (Figs. 9 and 10, loc. cit., plate II.). These strata, however, gave rise only to a very limited production of foreign elements, the main bulk springing from clusters situated in the inner granular layer.

How could the tumor, nevertheless, *occupy the vitreous space and be enveloped by the detached retina and pars ciliaris?* Since this forms the distinctive feature of our case, and has not yet been mentioned in literature, I will endeavor to render the explanation clearer by a diagrammatic illustration based on the foregoing examination. Fig. 6, Tab. II. represents an imaginary section through the globe, parallel to and a little behind the ora serrata, passing, therefore, through the centre of the pseudoplasm. *scl* indicates the sclerotic, *ch* the choroid, both healthy; *sr* the subretinal space, filled with serous fluid, as in an ordinary case of detachment of the retina. *r* denotes the wrinkled retina, enveloping the tumor *t;* between the two there is a space (*c*, Figs. 6 and 1) filled with fluid and being, as we have seen, a remnant of the vitreous chamber. The free surface of the tumor (*b*, Figs. 6 and 1) is smooth, and consists of the limitans *interna* and the adjacent *inner* strata of the retina; the outer strata, as above described (Fig. 4, Tab. III.), giving origin to the bulk of the tumor (*t*, Figs. 6 and 1). These are the positive data. We have, moreover, seen that, aside from the glioma, inflammatory changes were found in most parts of the retina. These

inflammatory changes may have been occasioned by the specific growth, but it is also conceivable that they were primary, and ushered in the gliomatous production. Be this as it may, this much is certain, that inflammation was present during the development of the growth, and we may fairly suppose that it caused a firmer adherence of the retina to the choroid than is usual. Glioma springing from the inner retinal strata will, as in Iwanoff's case, penetrate into the vitreous without loosening the connection between choroid and retina, but if it springs, as in our case, mainly from the outer layers, it will find an invincible resistance in the choroid and sclerotic. The growing mass, therefore, must crowd the limitans interna and adjoining retinal layers into the vitreous chamber. The more the tumor grows, the greater will be the tension of the inner retinal layers, and this cannot take place without exerting a traction on the adjacent portions of the retina, which then will become detached from the choroid and pushed inward into the vitreous. There is now a partial detachment of the retina, differing from ordinary detachment in that its cause is not subretinal fluid, but a growth which, situate in the outer retinal layers, acts like a subretinal tumor. The pseudoplasm, starting as it did from a point in the retina near the ora serrata, caused a traction which extended also to the pars ciliaris retinæ, and brought about its separation from the ciliary processes. After a portion of the retina was detached in this manner, and forced inward towards and perhaps beyond

the optical axis, the total detachment of the retina must have taken place. The dissolved vitreous fluid, partly by simple transfusion, partly in consequence of the contraction of the detached retina, exuded through this membrane and filled the space between retina and choroid. Thus the folded retina could envelop the glioma tumor more or less closely, and the puzzling conditions of this specimen, particularly the fact that the convex surface of the tumor consisted of the inner retinal layers, are satisfactorily accounted for.

In conclusion I may be allowed to make some *practical remarks* suggested by the foregoing case.

It is conceivable that the anterior portion of the suspensory ligament of the crystalline lens might also have given way during life. Then a dislocation of the lens into the vitreous chamber would have ensued, the peculiar origin of which must have rendered the diagnosis of the tumor very difficult.

A delay of the enucleation would have been fatal to the patient.

The *prognosis* of this case, quoad vitam, is certainly doubtful, considering the malignancy of retinal glioma. Life may, however, be preserved, since the pseudoplasm is still so confined, that, with the exception of the retina, no part of the eye is affected. The integrity of the optic nerve is of great importance.

The very early removal of the whole organ was, therefore, a step approaching the practical solution of the question on morbid growths in general, and on intraocular

tumors in particular, a question which, as a maxim, may be expressed thus: extirpate when the tumor is still a local disease, that is, before infection of the system has begun, and life will be saved.

EXPLANATION OF PLATES.

TABLE II.

Fig. 1. Meridional section through the eye, passing through the middle of the tumor.

t=tumor.

r=retina.

a=retina at the ora serrata.

pc=pars ciliaris retinæ.

v=vitreous space behind lens.

c=vitreous space between

b=free and smooth surface of the tumor and

e=detached retina.

d=vitreous space confined by folded and detached retina.

t_1=miliary retinal gliomata.

Fig. 2. Diffuse gliomatous degeneration of retina (at A), with infiltration of inner layers (B), and perforation of limitans interna.

b=small round cells.

c=small cells with shining nucleolus, like the nuclei of sarcoma cells.

g=choroidal pigment

a=blood-corpuscles in a blood-vessel.

d=white blood-corpuscles (lymphoid cells) lying between the fibres of the wall of the vessel.

e=perivascular space.

f=colloid bodies (myeline ?).

Fig. 3. Anterior portion of retina (*r*, *r*), ora serrata (*o*, *s*), and pars ciliaris retinæ (*pc*), enclosing glioma cells (*b*, b_1), fatty cells (*c*), and blood-vessels (*d*).

B=glioma cluster in inner granular layer, near (*d*) retinal vessel, extendin through the inner layers into the vitreous chamber (b_1), and invading also the outer granular layer.

Fig. 5. Remainder of vitreous body behind the lens, metamorphosed into connective tissue.

b=posterior capsule intact.

aa=newly formed connective tissue.

vv=blood-vessel, newly formed.

c=glioma tissue.

Fig. 6. Diagrammatic figure to explain the development and growth of the tumor.

scl=sclerotic.

ch=choroid.

r=retina.

sr=subretinal fluid.

gl, *t*=tumor.

r=retina, detached.

b=inner retinal layers pressed inwards by the tumor developing in the outer layers.

c=remnant of vitreous chamber between tumor and detached retina.

TABLE III.

Fig. 4. Glioma clusters in outer layers of retina.

li=limitans interna.

rf=radiating fibres.

n=nervous fibres.

g=ganglion cell.

le=limitans externa.

h=outer retinal layers, blended together, metamorphosed into connective tissue, and interspersed with granules.

ak=proliferation of outer granular layer.

gl=glioma cluster springing from inner granular layer, extending to outer granular layer (*ok*) and beyond it (*m*, *m*).

v=blood-vessel.

A CASE OF PTERYGIUM SUPERIUS.

By H. KNAPP.

THROUGH the kindness of Dr. C. F. Rodenstein, of Fordham, Physician to the Roman Catholic Protectory for Destitute Children, in Westchester County, New York, I had the opportunity of observing a case of this disease, and performing an operation for its cure. This affection is certainly very rare. For although I had been for years a regular student of some of the greatest eye hospitals in the world, and my own private and hospital practice furnished me with more than 22,000 recorded cases, I had never before seen a pterygium superius.

In the autumn of 1869, an epidemic of blennorrhoic conjunctivitis broke out in the above-named institution, which attacked over 300 out of the 1,500 children kept in this admirable charity. By the most energetic measures taken by Dr. Rodenstein as to treatment and cleanliness, and by the separation of the affected children, in which the Doctor received the efficient support of the Christian Brothers and Sisters of Charity in charge of the Institution, the epidemic was suppressed in six weeks, with no greater loss than that of four eyes.

Among the sequelæ of this form of conjunctivitis Dr. Rodenstein mentioned to me that two of the children suffered from pterygium superius, the one on one eye, the other on both. The former he brought to me for treatment. The patient was a girl of 11 years, whose right eye was healthy, but the left presented a well-developed, rather thick and vascular pterygium. On examination it was found that its basis was connected with, or rather sprang from, the whole superior reflection of the conjunctiva, while its apex overlapped the cornea so far as to cover the pupil entirely. (Fig. 8.) Grasping the false membrane with the teeth of an ordinary fixing forceps, I could raise it from the sclerotic, and pass a probe between it and the conjunctiva. The apex, however, was firmly adherent to nearly the whole extent of that part of the cornea which it covered. Holding the pterygium raised by the forceps, I detached it from the cornea with a cataract-knife, after which it retracted and lay stretched as a fleshy mass along the upper cul-de-sac, entirely hidden by the lid. A zone of healthy conjunctiva lying between the denuded corneal surface and the basis of the pterygium, I thought the latter, when left alone, would not unite again with the cornea, but disappear by shrinking. I therefore did not interfere with it any fur-

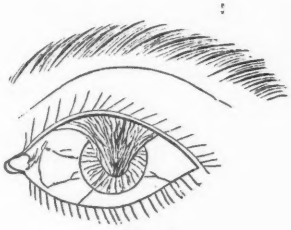


FIG. 8.

ther, and had the satisfaction of seeing my expectation fully realized. In about two weeks the pterygium had become almost invisible, and finally disappeared altogether. The eye itself showed a central *adherent leucoma*, which at first had been masked by the pterygium, and in which the whole inner half of the pupillary border was caught, while the pupil was hidden behind the opacity. I therefore performed an operation for artificial pupil, and restored a considerable amount of vision to the patient.

The *occurrence of pterygium superius* in consequence of blennorrhoic ophthalmia is mentioned by some of the earlier authors. Its mode of formation cannot, it seems to me, be doubtful. While the conjunctiva and cornea are ulcerated, the lids remain shut sometimes for days. During that time a union of the ulcerated surfaces takes place. The swollen upper reflection, lying in contact with the cornea, is very liable to be united with the corneal cicatrix. The conditions for the formation of such a union are certainly so frequent that the rarity of its occurrence is rather surprising. I presume, however, that this may be accounted for by the spontaneous detachment of many of such adhesions. In the majority of cases they will not be so extensive and of so peculiar a shape as to merit the name of pterygium, but are rather to be classified with simple symblephara partialia. And just as the hypertrophied portion of conjunctiva, in the case related above, shrank and disappeared after it had been separated from the cornea by the knife, so in

its less developed form will it shrink and disappear, when by the movement of the lids and globe a spontaneous detachment takes place. In the other case mentioned by Dr. Rodenstein, the pterygia had indeed disappeared spontaneously.

TUMORS OF THE RETINA.

By FRANCIS DELAFIELD, M.D., of NEW YORK.

(Plates IV. and V.)

The following cases of retinal tumors seem to be of sufficient interest to warrant their publication :—

Case I.—The eye was removed by Dr. H. D. Noyes. It was taken from a girl 18 months old. Her father had first noticed a white spot in the pupil of the eye three months before its removal. The child had no symptoms of a return of the disease for two months after the extirpation. After that time the patient did not return.

The eye, while still warm, was opened by a horizontal section. The cornea, iris, and lens preserved their normal appearance and position. The cavity of the globe, posterior to the lens, was filled with a soft, whitish mass, stained red in some places by hemorrhages, and so soft as to be semi-fluid.

The elements of this mass, examined without the addition of any fluid (Hartnack, No. 9, immers.), were round cells, of very pale, finely granular appearance, exhibiting no nucleus until iodized serum was added, and measuring ·0116 mm. Mixed with these cells were

amorphous granular matter, red blood-globules, and shrivelled round cells.

After hardening the eye in Müller's fluid, the tumor was seen to be continuous with the retina over about half its internal surface, and in apposition with it over the other half, the optic nerve being only covered by the tumor (Fig. 1). .

Sections were then made of the tumor and retina.

The retina, where it is only in contact with the tumor, is well preserved; the membrana limitans interna and radiating fibres are thickened, the other layers are unchanged. In some sections portions of the tumor remain adherent to the internal surface of the limitans interna. In sections of that part of the retina which is continuous with the tumor it is found that the rods and cones and membrana limitans externa are everywhere unaltered. In other sections the new growth reaches only to the external intergranular layer.

At the line where the tumor ceases to be continuous with the retina the growth of the tumor is seen to take place in two ways.

First: as we follow the layers of the retina towards the tumor, we see round cells making their appearance in the layer of nerve fibres, and a little farther on in the layer of inner granules, the internal intergranular layer remaining intact. These cells increase very rapidly in number, rendering the layers in which they occur much broader, pushing the membrana limitans interna inwards, pushing the external intergranular layer outwards, the

cells in the two layers coming in contact with each other, until there is one continuous mass of cells extending from the limitans externa inwards (Fig. 2).

Second: we see round cells making their appearance in the same layers, and increasing in the same way; but the limitans interna, instead of being pushed inwards, and then disappearing in the tumor, is pushed inwards and then turned over outwards against the adjoining portion of limitans.

The retina is not detached from the choroid. The choroid and optic nerve present no traces of new cells.

Robin, Manfrédi, and Iwanoff each describe a case of retinal tumor, in which the new growth was developed inwards. In all the other cases reported the tumors are said to grow outwards towards the choroid. In this case the growth was very markedly inwards, for while the tumor was of sufficient size to fill the globe, the bacillary layer was everywhere perfect and in contact with the choroid.

As regards the precise point in the retina which gave rise to the tumor, we can only say with certainty that it was to the inner side of the external intergranular layer.

Nearly all authors describe the cells which make up these retinal tumors as if they were identical with the granules of the retina, and formed by hypergenesis from them.

The supposition that they are formed by proliferation of the retinal granules seems to be purely hypothetical.

The idea that the cells forming the tumor are identical with the retinal granules seems to be due to the practice

of looking at specimens altered by preservative fluids with low magnifying powers.

The layer of outer granules is said by Schultze to consist entirely of cells connected with the rods and cones. The rod granules consist of a globular, homogeneous nucleus, with small shining nucleoli, and an extremely thin cortex of a pale cell-substance, which is distinctly visible only at the upper and lower ends of the nucleus, and from which fine fibres are given off. Henle, Schultze, and Ritter also describe a peculiar striation of these granules.

Henle describes the inner granules as of two kinds: (1), globular, transparent nuclei, measuring ·005 mm.; (2), the same nuclei enclosed by a scanty cell body.

The cells of this retinal tumor, examined fresh with No. 9 Hartnack, do not resemble any of these granules. On the other hand they resemble exactly the bodies commonly called lymphoid cells, which are found in so many new growths.

We can, therefore, leave on one side the question as to whether any of the retinal granules belong to the connective-tissue portion of the retina; for the elements of the tumor do not even resemble the granules, whatever the nature of the latter may be.

This case is one in which the prognosis should be favorable, as the disease was entirely confined to the retina. Unfortunately, the patient did not return after two months. For two months, however, there was no symptom of recurrence.

CASE II.—The eye was removed, by Dr. C. R. Agnew, from a child four years old. The disease of the eye was first noticed by the parents two years before its extirpation. During the last six months there was a great deal of pain in the eye, and the child's general condition became very bad. The child died within a year after the enucleation.

The eye was hardened in Müller's fluid. The cornea was transparent. The lens was pushed forwards, partly through the dilated pupil, into the anterior chamber.

The cavity of the globe was filled with a soft mass, mottled white and red. This mass was continuous with the optic nerve. Around the nerve the choroid was lifted up by a tumor of considerable thickness, and at the equator by another thinner tumor (Fig. 3).

The soft tumor, which fills the globe, evidently has its seat in the retina, leaving, however, no traces of the normal retinal tissue. The anterior portions of this tumor are composed of small round cells imbedded in an amorphous basement substance. The portion of the tumor near the optic nerve consists of larger cells, having a distinct cell body. These cells are contained in a delicate reticulum of connective tissue.

The retinal tumor is in contact with the entire internal surface of the choroid, and a thin white layer adheres in many places to the choroidal epithelium. The stroma of the choroid is thick and fibrous; the epithelium is thickened in irregular patches; many of the epithelial cells are greatly hypertrophied. In many places the epithelium is lifted up from the basement membrane by small, round cells, arranged either in little globular

masses, or forming a thin flat layer. Some of these globular masses of cells break through the epithelium, but none are seen to penetrate the basement membrane.

The stroma also is infiltrated with round cells, mostly arranged in long bundles. In the large tumors around the optic nerve there is nothing left of the choroid but its basement membrane and some irregular epithelium. There is, however, a distinct line of demarcation between the choroidal and retinal tumors. In all the subchoroidal tumors the cells have a distinct cell body, and are contained in a reticulum of connective tissue.

The optic nerve was cut off close to the globe. It is slightly enlarged, and the bundles of nerve fibres are replaced by round cells, the fibrous septa between the bundles remaining intact.

CASE III.—The eye was removed by Dr. Carmalt, from a girl 4 years old. Ten months before its removal the eye became red and painful, and soon after the pupil gave a greenish reflex. Six months before the removal of the eye there could be seen, with the ophthalmoscope, a tumor the size of a pea, in the outer portion of the fundus. Within four months after the extirpation a tumor appeared in the orbit, and a few months later the child died. A tumor was found in the brain at the optic chiasm.

The eye was hardened in Müller's fluid.

The cornea, iris, and lens are unchanged. The anterior chamber is filled with fibrine. There is an episcleral tumor extending from the optic nerve to just in front of the equator.

The cavity of the globe is nearly filled with a soft white tumor continuous with the optic nerve. No retina can be seen (Fig. 4). Around the nerve the choroid is lifted up by flat tumors, extending on one side as far forward as the ciliary body. There are the same changes in the epithelium as were described in the last case. The subchoroidal tumors do not break through the basement membrane, even around the optic nerve.

The optic nerve was cut off at a distance of 23 mm. from the sclera. It is uniformly increased in diameter. The nerve fibres have entirely disappeared and are replaced by round cells. The fibrous septa between the bundles of nerve fibres preserve their normal arrangement, and in a few places contain oval and round cells. The inner sheath of the nerve is thickened and contains a few round cells; the outer sheath appears normal. There are no cells between the two sheaths (Fig. 5).

The episcleral tumor has the same structure as the subchoroidal growths. There are several small aggregations of cells in the sclera between the choroidal and episcleral tumors, but there is nowhere any regular cell communication between the intra and extra ocular growths.

Case IV.—The eye was removed by Dr. Roosa, from a boy two years old. An opacity in the pupil of this eye was first noticed one year before its enucleation. Within four months the vision of the eye was destroyed, and the eye-ball began to protrude. A tumor situated in the upper and outer part of the orbit, and attached to the globe, was removed with it.

One month after the extirpation the tumor reappeared in the orbit. Soon after this the sight of the second eye was lost; there was hemiplegia of the left side, marked evidences of cerebral disease, and the child died in a short time.

The orbital tumor has the size of a pigeon's egg, is of ovoid shape, and is attached to the sclera.

The eyeball measures 30 mm. in its antero-posterior diameter, and between the tips of the ciliary processes 12 mm. There is an annular staphyloma in the sclera, between the corneal insertion of the iris and the ciliary body. The iris is carried forwards with the cornea.

The lens is transparent; it is held in place by the suspensory ligament, which is lengthened, thickened, and of a white color. The anterior surface of the lens is 2 mm. posterior to the plane of the iris.

There is a large white mass, extending like a pillar from the posterior surface of the lens backwards to the optic nerve. This mass does not touch the choroid, except at a point close to the nerve.

The internal surface of the choroid is covered by a thin white layer, composed of round cells. The epithelium is in many places lifted up by aggregations of round cells.

On the side of the nerve where the retinal tumor is in contact with the choroid, the latter is lifted up by a tumor reaching forwards as far as the equator.

The attachment of the episcleral tumor is over the sub-choroidal growth. The sclera between the two is thinned, and in some places infiltrated with round cells;

but neither the retinal, choroidal, or episcleral tumors are anywhere continuous with each other (Fig. 6).

The optic nerve is cut close to the globe, the nerve-fibres are replaced by round cells.

The new growth has the same characters as in the previous cases. The retinal tumor is composed of small round cells. The choroidal and episcleral tumors are composed of cells having a larger cell-body, and contained in a fibrous stroma.

CASE V.—The eye was removed by Dr. Mason from a girl 2½ years old. Six months before the operation it was first noticed that the pupil of the right eye was enlarged. When the eye was removed the orbit was scraped and washed with ac. acetic. Six weeks after the operation the tumor recurred in the orbit. Afterwards tumors appeared on the temples and inferior maxilla; the vision of the other eye was destroyed, and death followed three months after the operation.

The anterior two-thirds of the globe has its normal shape, the posterior third is continuous with an irregular mass of new tissue.

The cornea is opaque; the lens is pushed forwards against the posterior surface of the cornea.

The cavity of the globe is filled with a tumor, soft and white at its centre, firm and gray at its periphery. The choroid and retina cannot be distinguished (Fig. 7).

The sclera is unaltered over the anterior two-thirds of the globe, over the posterior third it is lost in the tumor.

The central soft portion of the tumor is composed of small round cells, the peripheric portion contains in

addition a fibrous stroma. Where the tumor has replaced the sclera, it can be seen that the latter has been split up into layers of fibrous tissue separated by cells.

The optic nerve is surrounded by the extra-ocular tumor. There are groups of round cells between its two sheaths, and replacing the bundles of nerve-fibres.

The extra-ocular tumor is continuous with the posterior portion of the globe. It is surrounded by thickened connective tissue and by the ocular muscles. The muscles are not invaded by the new growth.

Remarks.—The last four cases exhibit the methods in which the new growth spreads from the retina to the neighboring parts.

The choroid, optic nerve, episcleral tissue, sclera, brain, and periosteum of the cranial bones become the seats of secondary tumors.

The optic nerve was involved in four cases, and in all presented the same changes. The nerve was increased in size, but was contained in its sheaths. The two sheaths were very little altered in any of the cases, and in only one case were there cells between the two sheaths. The bundles of nerve-fibres disappeared, and were replaced by round cells. The fibrous septa remained unaltered. The cells formed a continuous growth with the retinal tumor. It will be noticed that this description differs from that given by most authors, for they describe the new growth as advancing along the fibrous septa and sheath of the nerve, leaving the nerve-bundles untouched until the last.

In the choroid the secondary tumors occur by discontinuous infection. Separate foci are formed beneath the epithelium and in the stroma. Those beneath the epithelium increase in size until they break through it, but were nowhere seen to break through the basement membrane. Even over the large sub-choroidal tumors around the nerve the same small sub-epithelial tumors existed. In addition there were irritative changes in the epithelium, causing hypertrophy of its cells.

The tumors in the stroma also increase in size, pushing the basement membrane inwards. They reach their greatest size around the nerve, and come closely in contact with the retinal tumor; even here, however, the basement membrane is not broken through. In Case V. we can no longer distinguish between retinal and choroidal tumors, but it seems probable that there were sub-choroidal tumors which broke through the basement membrane, and became continuous with the retinal tumor.

That these discontinuous choroidal tumors were produced by cells from the retinal tumor seems probable, but not certain.

The secondary tumors in the sclera were of small size, formed of aggregations of cells separating the fibrous tissue. This cell-growth reached its greatest development in Case V., where the sclera is split into many layers, so as to form a large mass continuous with the posterior part of the globe.

The tumors on the outside of the sclera reached in Cases IV. and V. a considerable size. In Cases III. and

IV. these tumors were completely separated from the intra-ocular growths.

In Case V. there were metastatic tumors growing from the periosteum of the cranial bones.

From the study of these cases I venture to draw the following conclusions :—

1. The rule laid down by Hirschberg, that retinal tumors grow outwards towards the choroid, has many exceptions.

2. The elements of these tumors only resemble the granules of the retina when altered by reagents and seen with low powers. When examined fresh and with high powers, they are seen to be identical with the so-called lymphoid cells which compose so many new growths.

3. If we dismiss from our minds the superficial resemblance between the elements of these tumors and the retinal granules, they at once take their place in the class of round-celled medullary sarcoma.

4. The variation in the size of the cell-body and in the proportion of stroma in different parts of these tumors, is almost always found. This variation is the same which we find in most sarcomata, and is no reason why we should speak of a tumor as beginning as a glioma, and then becoming sarcomatous.

5. Not only the anatomy, but also the clinical history of retinal tumors corresponds exactly with those of the medullary sarcomata.

6. The development of secondary tumors follows the rule laid down by Virchow for sarcomata, and occurs: (1) By continuous infection of the retina, optic nerve,

and, perhaps, the brain; (2) By discontinuous infection, forming the choroidal, scleral, and episcleral tumors; (3) By metastasis proper, forming tumors on the bones, lymphatic glands, and liver.

The idea that the choroidal tumors are formed by continuous infection from the retinal tumors, and that the scleral and episcleral tumors are formed by continuous infection from the choroidal tumors, seems to be erroneous.

In the very great majority of sections these tumors are evidently discontinuous. And, even if in some few instances a scanty cell communication is found between the two, it seems most probable, from what we know of other sarcomata, that this is a cell-growth posterior to the development of the secondary tumor.

That, however, these secondary tumors, although discontinuous, are produced from cells which have wandered from the parent tumor, is possible.

EXPLANATION OF TABLES IV. AND V.

FIG. 1.—Represents a horizontal section of the eye described in Case I. The tumor is seen to arise from about half the retina and fill the globe. The lens is pushed forwards.

FIG. 2.—Is a section of the retina at the point where it becomes continuous with the tumor, from Case I. The layers of inner granules and of nerve-fibres and ganglion cells become filled with round cells, and these cells become so numerous that they entirely replace all the layers of the retina as far as the limitans externa.

FIG. 3.—Is described in Case II. The cavity of the globe is filled with a tumor continuous with the optic nerve. On each side of the nerve the choroid is lifted up by tumors of some size.

FIG. 4.—Is described in Case III. The globe is filled with a tumor continuous with the nerve. On each side of the nerve the choroid is lifted up by small tumors. On the outside of the globe is a tumor of the same nature.

FIG. 5.—Is part of a cross-section of the nerve from Fig. 4. The neurilemma and fibrous septa are unaltered, while the nerve-fibres between them are replaced by round cells.

FIG. 6.—Is described in Case IV. The cornea and iris are carried forwards by an annular staphyloma, while the lens remains in place. A tumor, continuous with the nerve, extends from behind forwards through the centre of the globe. Beneath this the choroid is lifted up by a flat tumor, and the attachment of the episcleral tumor can be seen on the outside of the sclerotic.

FIG. 7.—Is described in Case V. The globe is entirely filled with the new growth, which has posteriorly split up and broken through the sclera so as to form a projecting tumor outside the globe.

FIGS. 2 and 5 were drawn from my preparations by Dr. Eno.

The other figures are from photographs of the specimens, by O. G. Mason.

CASE OF ANGIOMA CAVERNOSUM, SITUATED IN THE EXTERNAL AUDITORY CANAL, AND ATTACHED BY A SLENDER PEDICLE TO THE MANUBRIUM MALLEI.

REPORTED BY ALBERT H. BUCK, M.D.,
Assistant Aural Surgeon to the New York Eye and Ear Infirmary.

(PLATE VI.)

George Algerer, æt. 19, mechanic. Admitted to the New York Eye and Ear Infirmary, April 2d, 1870.

History.—When seven years old, patient had some acute sickness, either measles or scarlet fever, after which he began to suffer from earache and a purulent discharge from both ears. Since that time the discharge has continued almost uninterruptedly, and has been accompanied by occasional attacks of earache. Otherwise his health has been tolerably good. There has never been any blood in the discharge, but on one or two occasions he has noticed a little blood on his finger after pushing it rather too violently into the ear. As far back as he can remember he has been troubled with either a ringing, buzzing, or whistling noise in both ears; at certain times the sound was like that of a steam-whistle. Of late the sound in the left ear has been what might be termed a pulsating humming noise; it is continuous, but at regular intervals, corresponding apparently to the pulse-beat, it becomes louder. Running up stairs, or any other violent exercise, aggravates very much the noise.

On waking this morning he found his pillow, as he states, covered with blood. The hemorrhage came from the left ear, and continued, in the form of oozing, throughout the entire forenoon. He suffered no pain, and could assign no cause for the occurrence.

Present Condition.—Patient's general condition is good. He can only understand very loud conversation. On the right side a loud-ticking watch cannot be heard at all; on the left he hears it when pressed against the concha. Right meatus half filled with pus, obscuring the field of vision. After its removal the membrana tympani was found to be entirely destroyed, and its place occupied by granulations springing from the internal wall of the tympanum. The short process of the hammer and its apparently shortened handle were yet visible. Left meatus filled with blood, partly clotted. After its removal by syringing, an oblong, dark-colored body, about the size of a small pea, was found lying apparently free on the inferior wall of the meatus. At first sight it was supposed to be a foreign body, or inspissated cerumen. It was only after several vain attempts with the syringe that the small body was discovered to be attached by a long and slender pedicle to the stump of the hammer, which could be seen in the background. Dr. Robert F. Weir, to whom I am indebted for the case, then divided the pedicle with the scissors, after which the growth was readily removed by syringing. No appreciable hemorrhage followed. Ordered insufflation of powdered alum.

April 23d. No discharge from either ear at present. Hearing distance remains the same. The noises previously complained of have ceased. On the left side the yellowish-white stump of the hammer can be seen standing out rather prominently from the upper portion of a red and slightly granular field. The membrana tympani is entirely gone; no trace can be seen of the former pedicle.

June 25th. Patient returned to-day with the statement that soon after the last visit the noises and discharge both reappeared, and have continued ever since. Right and left meatus are both filled with pus. After a thorough cleansing the appearance of the left ear is found to be the same as described above, with the exception that now a pinkish

vascular teat hangs from the stump of the hammer. Its bright color is in decided contrast with the granulations surrounding it on almost every side. Ordered instillation twice daily of

℞. Acidi tannici ..... ʒ i.
Glycerinæ ..... ℥ i. M.

Microscopic Examination.—Immediately after its removal the small growth was placed in a weak solution of chromic acid—the only preservative fluid which happened to be at hand at the time. From this it was transferred to alcohol, and when sufficiently hardened it was mounted in a mixture of wax and oil, in equal parts. The sections obtained with the razor were first tinged with carmine, and then mounted permanently in Damar varnish. From a study of these sections I am enabled to give the following description:—

The surface of the tumor is covered by a moderately thick fibrous membrane, intimately connected with the tissues beneath it. The central portion is an irregularly-shaped cavity, filled with blood. The remainder of the growth presents everywhere the same appearances, namely, sections of blood-vessels of different sizes and shapes, and a fine intervascular network of connective tissue, containing blood-corpuscles in its meshes. Toward the periphery the vessels are arranged side by side, as if radiating from the centre of the growth. The smallest of these measured mm. 0.024, the largest mm. 0.324. Immediately beneath the capsule the walls of some of the vessels measured as much as mm. 0.02 in thickness. No capillaries can be found in any part of the growth.

In the neighborhood of the central cavity there are seen sections of what seem to be irregularly-distended veins. In a few instances these can be seen terminating directly in the central cavity; in others, one or more of the radiating vessels can be followed into the large vein. In many of the vessels the calibre is traversed by a network of fine threads of connective tissue, holding blood-corpuscles in its meshes, and differing in no respect from the network located between the vessels (Fig. 2). Where the section is sufficiently thin, a few scattered white corpuscles can be distinguished among the red.

The point of rupture can easily be recognized in some of the middle sections by the presence of a plug of clotted blood extending from the central cavity to the periphery of the tumor (Fig. 1).

In the stump of the pedicle only one large vessel can be distinctly recognized. It becomes forked very soon after its entrance into the tumor, and one of the branches can be traced directly into the central cavity.

Remarks.—The question which naturally presents itself after an examination of such a vascular growth is: —are we to consider it as a form of fibroma rich in blood-vessels, or as a true angioma?

The most recent articles on the subject of aural polypi are those of Kessel and Steudener, published in Vol. 4 of the *Archiv für Ohrenheilkunde.* These writers give the reports of the microscopical examination of 75 aural polypi made by Tröltsch (3), Meissner (5), Förster (5), Billroth (7), Kessel (22), and Steudener (33).

Among Kessel's 22 polypi he reports one, "the greater portion of which consisted of blood-vessels and corpuscles. Near the surface the vessels were purely capillary, consisting entirely of nucleated protoplasma cells. . . . . From what has been said above, we must consider this as a coagulum of blood in process of organization. The growth is therefore analogous to the fibrinous blood polypi which occur in the uterus."

Steudener, speaking of mucous polypi of the ear, says: "In many cases the vessels are found in a state of extreme dilatation, and with extraordinarily thin walls; often at the same time they are so numerous that sections through such polypi remind one of vascular tumors."

These are the only references I can find to aural polypi resembling in the slightest degree the growth which I have attempted to describe above.

Virchow, in his admirable work on tumors, treats the subject of angioma with great clearness and fulness. It is from a careful study of this account, and from the confirmation of Drs. Knapp and Francis Delafield, that I have ventured to classify this growth as a *polypoid angioma cavernosum.*

Virchow defines an angioma to be a tumor composed chiefly of newly-formed vessels, or of vessels in whose walls there are newly-formed elements. He applies the term *cavernosum* to those angiomata which, in the place of capillary blood-vessels, possess blood spaces. In such growths, he continues, the arteries pour their blood im-

mediately into these spaces, from which it then passes into the veins, and so back into the general circulation.

In the present case we have a distinct polypoid tumor, composed chiefly of blood-vessels and blood-containing spaces. In the centre is a cavity communicating not only with some of the large veins, but also with the blood spaces; and from it at least one large vein passes into the pedicle. The circulation must probably have taken place after this wise:—From the arteries the blood passed into the spaces of the spongy tissue, and thence either directly into the smaller veins, and so on into the large vein of the pedicle; or first into the central cavity, and then into the general circulation.

As the patient was sleeping quietly on the affected side at the time of the hemorrhage, the accident was probably due to causes having their seat within the tumor itself. Pressure, progressive atrophy, and finally rupture, through simple weakness of the tissues to resist the ordinary force of the heart, may safely be assumed as the cause of the accident.

Finally, the reappearance, some six weeks after the operation, of a highly vascularized tissue at the spot previously occupied by the pedicle of the polypus, may be reasonably looked upon as a confirmation of Virchow's view, that an angioma is a true vascular hyperplasia.

EXPLANATION OF DRAWINGS.

FIG. 1.—Section through entire polypus. At *d* can be seen the stump of the pedicle, including in its substance the efferent vein *e*. At *g* the rupture occurred. The central cavity *f* communicates both with the rupture *g* and the vein *e*. For the sake of clearness, the clotted blood, which filled all these spaces, is left out of the drawing. At *c* is represented a separate outgrowth, resembling in its composition the tissue of the main tumor. Sections of blood-vessels can be seen at *b* and elsewhere. The entire mass is surrounded by a capsule *a*. 20 diameters.

FIG. 2.—Section through the spongy tissue of the polypus. At *a* is represented the connective-tissue network, containing in its meshes blood-corpuscles *b*. The white corpuscles, which are more or less tinged with carmine, are drawn at *c*. 300 diameters.

SPECIAL APPLICATION TO THE CASE OF THE EYE OF KNAPP'S GENERAL FORMULÆ FOR ASTIGMATIC RAYS.

(*See A. F. O., Vol. 8, Part 2, pp.* 198–201.)

By Dr. G. HAY, of Boston.

Prof. Knapp, following Kirchhoff, has shown that a small pencil of astigmatic rays may be considered, under certain suppositions,* as passing, not through a single point, as in the case of homocentric rays, but through two straight lines, each perpendicular to the direction of the axis of the pencil, and at a greater or less distance from each other; that is, that each ray of the pencil intersects each of the two straight lines.

Prof. K. shows that in the human astigmatic eye these

* That this is true only under certain suppositions, appears not only from Knapp's analysis, but also from the fact that we can conceive of pencils of rays in which it is not the case; as, for instance, a pencil of normals to the surface of the right cone, where the axis of the cone, which is intersected by all the normals, is not perpendicular to any of them. See also in the Transactions of the Am. Ophthalmolog. Soc. for 1866, the form of an astigmatic pencil resulting from the refraction of a small oblique homocentric pencil, by a glass plate, with plane parallel surfaces, where one of the focal lines is straight and oblique to the axis of the pencil, the other being curved, and at its intersection with the axis of the pencil, perpendicular to this axis.

two focal lines are at right angles to each other (though not intersecting), as well as each at right angles to the direction of the axis of the pencil; and it is on this supposition that our conception of the corrective action of cylindrical glasses is based; inasmuch as by means of a simple cylindrical lens, with its cylinder-axis parallel to one of the focal lines, we can, without materially altering the position of the other, bring up the first to the second, so as to intersect each other; and then all the rays, having to pass through each of the focal lines, must pass through their intersection, and so become homocentric.

The mathematical development given by Knapp does not appear to be limited to this one supposition,* the

* For if we regard the equations merely algebraically, and put A = 1, B = 1, C = 2, and D = 1, then Knapp's eq. (III.)

$$[1 + z(A - 1)][1 + z(D - 1)] - z^2 BC = 0 \quad \text{(III.)}$$

will be satisfied by the two real values

$$z = \sqrt{\tfrac{1}{2}}$$

$$\text{and } z = -\sqrt{\tfrac{1}{2}}.$$

The former of these, $z = \sqrt{\tfrac{1}{2}}$, substituted in either of eqs. (IV.)

$$\left.\begin{aligned} \frac{y}{x} &= \frac{1 + z(D - 1)}{zB} \\ \frac{y}{x} &= \frac{zC}{1 + z(A - 1)} \end{aligned}\right\} \text{(IV.)}$$

$$\text{gives } \frac{y}{x} = \sqrt{2},$$

and the latter, $z = -\sqrt{\tfrac{1}{2}}$, substituted in either of eqs. (IV.) gives

$$\frac{y}{x} = -\sqrt{2}.$$

Consequently the two corresponding focal lines would not be at right angles to each other.

It should be mentioned that eqs. (IV.), which are dependent on the supposition of eq. (III.), indicate the direction of a line perpendicular to the axis Z, and which

one with which we are most concerned in applying glasses. It is desirable to limit the general development by such conditions that it shall represent the special class of cases which we have to treat in the eye.

If we inquire into the meaning of the co-efficients A, B, C, D, used by Knapp, we shall find that the conditions of astigmatism of the cornea may be introduced by putting B and C each equal to zero.

This may be seen as follows: Knapp's method contemplates the position of the points in which any one of the rays intersects certain planes at right angles to the axis of the pencil. The rays are referred according to the methods of analytical geometry to three rectangular axes X, Y, Z. One of the rays is made the co-ordinate axis Z, and this may be taken as the axis of the pencil.

x_0, y_0, z_0 ($= 0$) represent the co-ordinates of the point in which a ray intersects the co-ordinate plane XY.

x, y, z represent the co-ordinates of the point in which the same ray intersects a plane perpendicular to the co-ordinate axis Z, at distance z from the origin.

x_1, y_1, z_1 ($= 1$) represent the co-ordinates of the point in which the same ray intersects a plane perpendicular to the axis Z, and at the distance unity from the origin.

Knapp makes x_1 and y_1 each dependent on x_0 and y_0, (these latter being supposed quite small); thus

$$x_1 = Ax_0 + By_0$$
$$y_1 = Cx_0 + Dy_0$$

all the lines of the pencil intersect. As eq. (III.) is satisfied by two values of z, there may be two focal lines determined by eqs. (IV.), both of which equations hold for each of the two focal lines.

If we compare these equations with the formula for the development of a function (x_1) dependent on two variables (x_0 and y_0), it will be seen that

A represents $\frac{dx_1}{dx_0}$

B " $\frac{dx_1}{dy_0}$

C " $\frac{dy_1}{dx_0}$

D " $\frac{dy_1}{dy_0}$

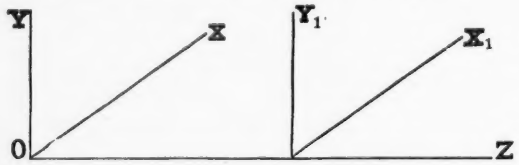
The several differential co-efficients being taken at the point for which

$$x_0 = 0$$

and $y_0 = 0$.

We have supposed the axis Z to be one of the rays and the axis of the pencil.

Now (remembering that in the analysis we merely contemplate the position of the rays without considering how they get their position), if we put $B = \frac{dx_1}{dy_0} = 0$, it will mean that (see Fig.) a ray meeting the co-ordinate



plane YX in the co-ordinate axis Y, and at a short, strictly speaking, an infinitesimal, distance from the origin O, will intersect the plane Y_1X_1 in the line Y_1, that

is, will lie in the plane YZ (Y_1 being parallel to Y, and X_1 to X), will not deviate to one side or the other of this plane YZ, though it may be inclined to or from the axis Z.

Likewise, if $C = \frac{dy_1}{dx_0} = 0$, it will mean that a ray meeting the plane YX in the axis X, and at an infinitesimal distance from 0, will intersect plane Y_1X_1 in line X_1, that is, will lie in plane ZX, will not deviate from this plane, though it may be inclined to or from the axis Z.

This is what we have in the eye: rays proceeding from a point in the optic axis, and meeting the cornea at the principal meridians, remain in the planes of these meridians after refraction by the cornea; these planes being perpendicular to each other.

If this arrangement is disturbed by the crystalline, we suppose, with a view to treatment, that the resultant action of the cornea and crystalline gives an astigmatic pencil of the same kind as that given by the astigmatic cornea.

With this explanation of the meaning of the conditions $B = 0$, and $C = 0$, we proceed to inquire what form Knapp's equations will take under such limitations.

Knapp's eqs. (1) and (2) must hold for the whole length of each of the lines of the pencil. Now, for two special values of z, the second member of (1) and of (2) vanishes. The first member of (1) equals zero for each of these two values of z; and also for each of these same two values the first member of (2) equals zero. That is,

$$x\,[1 + z\,(D - 1)\,] - y\,z\,B = o, \quad [1]^{I}$$
$$y\,[1 + z\,(A - 1)\,] - x\,z\,C = o, \quad [2]^{I}$$

and the two values of z, for which eqs. $[1]^{I}$ and $[2]^{I}$ hold, are the two roots of eq. (III.).

Now, it is proposed to consider the cases in which

$$B = o$$
$$\text{and } C = o$$

Then $[1]^{I}$ and $[2]^{I}$ become

$$x\,[1 + z\,(D - 1)\,] = o \quad [1]^{II}$$
$$y\,[1 + z\,(A - 1)\,] = o \quad [2]^{II}$$

and the two values of z, for which $[1]^{II}$ and $[2]^{II}$ hold, are those which satisfy eq. (III.) after putting $B = o$ and $C = o$, that is,

$$[1 + z\,(A - 1)]\,[1 + z\,(D - 1)] = o$$

These values are—

$$z = -\frac{1}{A - 1} \quad [3]$$

$$\text{or } z' = -\frac{1}{D - 1} \quad [4]$$

Now $[1]^{II}$ and $[2]^{II}$ hold each for [3] and for [4].

§ 1. Substitute [3] in $[1]^{II}$ and in $[2]^{II}$ and they become

$$x\left[1 - \frac{1}{A - 1}(D - 1)\right] = o \quad [1]^{III}$$

$$y\left[1 - \frac{1}{A - 1}(A - 1)\right] = o \quad [2]^{III}$$

$[1]^{III}$ gives $x = o$

$[2]^{III}$ gives $y =$ undetermined.

Therefore at the distance $z = -\frac{1}{A-1}$,

$$y \text{ is undetermined}$$
$$\text{and } x = o.$$

These co-ordinates are for one point in each of the lines of the pencil. Each of the lines passes through a point corresponding to these co-ordinates. The locus of such points is a straight line in co-ordinate plane ZY, and perpendicular to the axis Z.

§ 2. Substitute [4] in $[1]^{II}$ and in $[2]^{II}$ and they become

$$x\left[1 - \frac{1}{D-1}(D-1)\right] = o \quad [1]^{IV}$$

$$y\left[1 - \frac{1}{D-1}(A-1)\right] = o \quad [2]^{IV}$$

$[2]^{IV}$ gives $y = o$

$[1]^{IV}$ gives $x =$ undetermined.

Therefore at the distance $z' = -\frac{1}{D-1}$

$y = o$

and x is undetermined

These co-ordinates belong to a point in each line of the pencil. Each of the lines passes through a point corresponding to these co-ordinates. The locus of these points is a straight line in the co-ordinate plane XZ, and perpendicular to the axis Z.

Thus the two focal lines are perpendicular to each other, though not intersecting each other.

Thus, under the above suppositions, we have two principal planes, the co-ordinate planes XZ and YZ, at right angles to each other; and in each of these, perpendicular to their common intersection, a focal line, the two focal lines, however, not intersecting each other.

Now each ray of the small astigmatic pencil intersects each of the two focal lines.

Therefore each focal line, being in one principal plane and perpendicular to the other, must pass through the focus of the rays in the other principal plane, otherwise these rays could not all intersect the focal line.

IRIDECTOMIA WITHOUT DIVISION OF THE SPHINCTER PUPILLÆ.

By B. A. POPE, of New Orleans.

EVERY ophthalmic surgeon must have been impressed by the imperfect *optical* results obtained in some cases of *corneal changes* where the operation for iridectomia, with division of the sphincter pupillæ, has been well performed.

The unsatisfactory optical result in such cases is *mainly* to be attributed to the action of one or more of the following conditions of the cornea, whose increased influence, in consequence of the enlargement of the pupil, may not be sufficiently counterbalanced by the beneficial effect of any more or less transparent cornea which may be found in front of the new pupil.

A. The whole thickness of the cornea may have been involved in diseased processes which have resulted in the most varied changes in its curvature. B. There may be superficial irregularities of the external surface of the cornea (facets, etc. etc.). C. The various forms of corneal opacity.

The unfavorable optical influence exerted *exclusively*

by the enlargement of the pupil towards the corneal margin is, as a rule, of small importance compared with that exerted by the corneal changes just mentioned.

In those cases where the above-mentioned corneal changes may be present, the preservation of the sphincter pupillæ is of peculiar importance. The enlargement of the pupillary space consequent upon the excision of a portion of the iris with division of its sphincter is not to be measured by the surface covered by the excised portion before the operation. To this must be added the dilatation of the remains of the sphincter pupillæ, the separation of the cut edges of the iris by the remaining radial fibres, and the retraction of the divided radial fibres towards their ciliary attachments.

Critchet's operation for displacing the pupil has replaced in many cases that for iridectomia; still there remain cases in which the operation for displacement is contra-indicated. For meeting the indications in these cases, I propose the operation for excision of a portion of the iris, without dividing the *sphincter pupillæ*. By this operation the *therapeutic* effects of the usual operation are, in part at least, retained, while the *optical* defects are much diminished. As compared with the operation for displacing the pupil, it is in most cases optically inferior in its results; but it avoids all of its dangers in those cases where the internal membranes of the eye may be diseased, or where the iris may be placed in mechanical conditions rendering displacement of the pupil dangerous or impossible.

As yet I have only operated upon six (6) cases after this method. The result in these cases has been quite satisfactory.

In no case has there been complaint of an unpleasant optical effect resulting from the double pupil.

For making the corneal incision, I have used a rather long and very narrow lance-shaped knife. As a rule, the incision should be made at the sclero-corneal junction; but if a very eccentric pupil has to be made, the incision should be in the sclerotic coat. If in a case the pupil has been much displaced laterally, as in perforation of the cornea near its margin, with prolapsus of the iris, it may be best to make the incision in the transparent cornea. The incision being made, the knife should be withdrawn without allowing the escape of the aqueous humor. The aqueous humor should be gradually evacuated, prolapsus of the iris being carefully guarded against. A fine curved pair of iris forceps should be used, the curve of the blades, however, being moderate, as it is particularly desirable only to seize the iris with the *teeth* of the forceps. The point at which the iris should be seized should be immediately behind that portion of the cornea most available for optical purposes, and only enough of its tissue should be seized to insure a firm hold. If the corneal wound be small, that portion of the iris between the periphery and the pupillary margin will in the act of withdrawal of the forceps be somewhat stretched, and thus favor the avoidance of a division of the sphincter. For excising the iris, very

delicate curved scissors should be used. The operator should carefully watch the pupillary margin as the forceps is withdrawn, and the excision should be performed as soon as the blades of the scissors can be passed between the cornea and the point of the forceps. In cutting the iris the scissors may be held at right angles to the direction of the radial fibres, or parallel to their course. In the first case the pupil will approximate more or less to the circular form, while in the latter it will be elongated in the direction of the radial fibres. The size of the pupil will be very much what the operator wishes it to be, after he has had some little experience in the performance of the operation. It would probably be best for the operator both to seize and excise the iris, but I have latterly trusted the excision to a competent assistant.

With one exception chloroform was used, and its use is advisable in all cases where there is any doubt as to the perfect steadiness of the patient.

If in any case the sphincter pupillæ should be unintentionally divided, the worst result would only be the performance of the old operation, with the smallest possible loss of iris tissue.

The disagreeable appearance of an eye operated upon in the usual way is in many cases a serious objection. When the sphincter pupillæ is not divided, and the new pupil has been made very small, this objection holds in a lesser degree. The pupil may be made smaller than the old one, and very nearly round, the cut extremities

of the radial fibres being still visible, instead of being lost behind the corneal margin, as is usual in the old operation.

Even in cases where the old operation gives its best *optical* results, this operation, and that for displacement, would give better. The practical tendency would therefore be to confine the application of the old operation to cases demanding a purely *therapeutic effect*, or a *combination* of *therapeutic* with *optical* results.

The operation downwards and inwards is the best, if the case admits of it. If the operation be made in the lower half of the iris, the patient can in many cases readily cover the old pupil by allowing the upper lid to droop, which is an indirect optical advantage of importance.

Upon a nearer examination of the cases in which I have operated after the new method, the four following classes may be formed :—

1. Those in which there is a central opacity of the cornea, and in which there has been acute or chronic internal inflammation of the eye, with or without adhesions of the iris to the lens capsule.

2. Those in which there has been perforation of the cornea with prolapsus of the iris, the pupil having suffered displacement, and being totally or almost entirely placed behind more or less opaque cornea. In case the cornea has suffered very decided changes in form, not amounting, however, to staphyloma, the operation is most strongly indicated. If there be present in the case iritis, or irido-choroiditis, which does not yield to treatment, it

may be best to operate with division of the sphincter. If the pupil has been drawn to a very eccentric portion of the cornea by the prolapsus of the iris, it may still be best, in some cases, to excise the iris rather largely, without dividing its sphincter. Where the whole pupillary margin has been engaged in the corneal opening, the operation for displacement, and that for iridectomia, after the usual method, are of course excluded.

3. Cases where there is diffuse opacity of the cornea, most marked centrally, and gradually diminishing towards its circumference, together with iritis or iridochoroiditis, with or without adhesions of the pupillary margin to the lens capsule.

4. Cases where an extremely eccentric pupil may be required and internal disease of the eye may exist. It may even be best in this class of cases when no actual disease may be present, but where there may be doubt as to whether the iris will allow of extreme displacement of the pupil without inflammatory reaction. This condition might be found in the cases of old persons in whom the pupil was rigid and but slightly dilatable by the action of atropine.

V. Graefe has recommended the use of the extract of calabar-bean in order to contract the pupil preparatory to the operation for iridectomia, in those cases of glaucoma where the pupil is evidently dilated. It may be, in some cases, beneficial in the proposed operation where the pupil is large and the pupillary margin free; but I

have as yet operated on no case in which its use was necessary.

Where a very small portion of the iris is excised without dividing the sphincter, the pupillary margin being free, the form of the old pupil undergoes but little change; but should the excision be made larger, the pupillary margin next to the cut radial fibres falls nearer to the centre of the pupil, and may approximate to a straight line. At the same time, the radial fibres next those cut draw the pupillary margin a little without the original circle, and thus assist in straightening that portion of the sphincter separated from the radial fibres.

In proposing this operation it is done with the full consciousness that a more extended experience is necessary, in order to determine with accuracy its relative merits. That in a few unpromising cases it has given excellent results, gives reason to hope that it may add something to our surgical resources in that class of cases for which it is proposed.

The germs of the operation found in the writings of the elder Wentzel and of Beer hardly need special mention here. The operation of Wentzel was in reality only applicable to certain cases before or after cataract extraction, and has been replaced by much better methods. Beer proposed the excision without touching the pupillary margin in cases where the iris was attached to the lens capsule. This he did on account of the supposed dangers of operative interference with the attached portion of the iris. This fear has been shown by subse-

quent experience to have been to a great extent groundless. It was not the *object* of either operator to gain the *best optical* result, but to make a pupil with the greatest ease and least risk possible, and this in cases in which the *cornea might or might not* be diseased.

THE LIGHT-STREAK SEEN IN THE CENTRE OF THE RETINAL VESSELS WITH THE OPHTHALMOSCOPE.

By EDWARD G. LORING, M.D., New York.

Shortly after the invention of the ophthalmoscope, Helmholtz,* in his description of the instrument, advanced the statement that the arteries of the retina were to be distinguished from the veins by their double contour, which was visible under the ophthalmoscope.

A little later, Van Trigt† showed that this was not the case, and pointed out that what Helmholtz had mistaken for the double contour of the arteries was in all probability the brilliant light-streak occupying the centre of the vessel, which was common also, though in a less degree, to the veins. Van Trigt was the first to ascribe this light-streak to the reflexion back towards the observer of those rays of light which under the ophthalmoscope struck upon the anterior surface (Vorderfläche) of the arteries.

* Beschreibung eines Augenspiegels. Berlin, 1851, p. 34.

† Dissertatio Ophthalmologica inauguralis de speculo oculi. Utrecht, 1853

Jaeger, in an article * written in 1854, accepts this explanation, and is particular to give a full and detailed account of the phenomenon; and as this account has been accepted by all the leading observers as the correct one, and as it forms the basis of the following remarks, I will give it in Jaeger's own words (p. 328, loc. cit.) :—

"The lighter red and more brilliant color of the arteries correspond to their clearer blood as well as to the stronger light-reflexion from their walls. The lighter and clearer centre of the arterial as well as venous vessels in its entire extent (on account of which they, at that place, appear more transparent, as if seen by transmitted light—and on account of which they appear to have on each side a single dark contour) is in fact only produced by increased reflexion of the rays from the surface of the vessels; or in other words, those rays which fall on the summit of the curvature of the vessel (relative to the axis of vision) are mostly reflected back in the direction of the rays entering the eye, while, on the contrary, the rays which fall upon the descending portions of the curved surface of the vessel are for the most part reflected in a sidewise direction.

"The vessels are, in fact, in their entire breadth, of a perfectly uniform color, and allow no light to pass through them; for which view not only do the extent of their diameter, clear centre, and dark contours speak, but what would also follow from this, namely, that the

* Ergebnisse der Untersuchung d. mensch. Aug. m. d. Augenspiegel. Vorgelegt in der Sitzung vom 27 April, 1854.

vessels, precisely there where they run perpendicular to the observer's axis of vision, show a clear centre, but lose the same when they run in a direction relatively oblique to this; that, further, the lighter artery, when it passes over the darker vein, shows, at the point of crossing, the same color and clear centre that it does when the vessel runs over the clear fundus; that, further, the clear centre, with slight displacement of the ophthalmoscope, also is displaced sidewise; while by changing the manner of illumination (as by using a mirror with a short focus) the light-streak fades away entirely."

Mauthner also, in his recent work on the ophthalmoscope (1867), accepts Van Trigt and Jaeger's explanation of the phenomenon, and distinctly states that "since the wall of the vessels (Gefässwandung) reflects sufficient light to make itself independently visible, so must the light-streak be the expression of this light-reflection." *

Jaeger, however, in his last work,† published in 1869, though he still adheres to his former explanation, and refers the reader to the passage just quoted, adds the following note:—

"The central vessels, when seen by the ophthalmoscope, appear as if bordered on each side by a single contour, while, however, under the microscope they show a double one. This difference is due to the fact, that the walls of the vessels and the retina are very nearly equally transparent, so that with the ophthalmoscope, under physiological conditions, it is only the red blood-columns, and not the walls of the vessels, that are seen; consequently the vessels are not perceptible in their entire

* Lehbruch der Ophthalmoscopie, Ab. 1, p. 244.

† Ophthalmoskopischer Hand-Atlas, 1869, p. 32.

diameter. The red blood-column reflects back towards the mirror the greater part of those rays of light which are thrown by the ophthalmoscope on its central portion, which therefore appears illuminated and brilliant; while, on the other hand, the lateral portions reflect the light falling upon them mostly in a sidewise direction, and consequently appear dark; they are, in fact, only apparent to the observer through diffuse reflected light. The single dark contour of the central vessels under the ophthalmoscope marks consequently the limit on either side of the red-colored portion of the blood-column, while, on the contrary, the double contour revealed on each side of the vessel by the microscope marks the outer and inner limit of the walls of the vessels themselves. Consequently, in regard to the central vessels, so far as the ophthalmoscope is concerned, the expression 'vessel' is, strictly speaking, incorrect, and should rather be 'blood-column.' The other vessels seen in the fundus of the eye, especially those of the choroid, possess walls which are so little transparent that they are visible as such for their entire diameter, and consequently their red blood-column is not independently recognizable."

It will thus be seen that Jaeger gives up the idea that the light is reflected back from the *walls* themselves, for the reason that they are too transparent to produce sufficient reflection. And in this I cannot but believe he is right, from the fact that the illuminated streak is present in some of the smaller vessels, the walls of which are perfectly transparent, and which, when emptied of their contents, are invisible under the ophthalmoscope. In the second place, even at the nerve and its immediate neighborhood, where the walls of the vessels are less delicate, and consequently less transparent, the light-streak is not invariably present, as it would be were it

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simply the effect of reflection of light from the vessel-wall itself.

Eliminating, therefore, the vessel-wall as a factor to any perceptible degree in the production of the light-streak, there still remain two direct statements which appear to me to be unfounded, notwithstanding the high authority from which they come. These are—

1. That the reflection takes place from the red blood-column.

2. That no light traverses the vessel.

I believe, in fact, just the opposite: namely, that the light striking the wall nearest the observer passes through this, on account of its transparency, without being reflected to any appreciable degree; traverses the contents of the vessel, and is then reflected back, slightly perhaps from the opposite wall, but principally from the subjacent tissues.

My belief in this is founded on the following facts:

In the first place, the light-streak is too broad to be a simple reflection from the vertex of a curved surface so small as the retinal vessel, be it either from the wall or blood-column. Secondly, a column of blood of these dimensions is too transparent, and of too unreflecting a nature, to give so brilliant a streak.

Again, if it were true that the light was reflected back from the blood-column, then it would follow that the light-streak would always be present as long as the vessels remained full, and the fuller they were, and consequently the more dense the column of blood, the

greater the reflection. That this is not the case is proved by the following facts:—

In normal eyes, in which there is a physiological venous pulse, the double contour is seen alternately to disappear and appear again with the rise and fall of the current. This effect, as is known, can be very much increased by exerting pressure externally upon the globe.

When this is done the double contour is seen to disappear along the vein *pari passu* with the pressure, till the light-streak becomes obliterated as far as the sight reaches, and immediately reappears as soon as the pressure is removed. The reason of this is evident. The venous pulsation is simply a slight temporary stasis of that portion of the venous current just prior to its exit from the eye. During this stasis the red globules are for a little space crowded momentarily together; this increases the opaqueness of the blood at that point, and the light, instead of passing through to the wall beyond and subjacent tissues, is so much absorbed that too little is reflected back to the observer's eye to give the effect of the light-streak. Now, what takes place at a single point with the natural venous pulse, also takes place along a large tract of the vessel when the stasis in the vessel is increased or made complete by artificial external pressure. It may be said, however, that by applying external pressure we so flatten down the vessel that it loses its cylindrical form, and thus the light is not then reflected directly back from the summit of its curvature, but is diffused across the vessel's entire diameter.

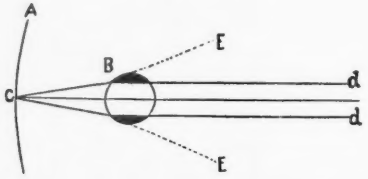
That this is not so is proved by the fact that, at the very moment that the light-streak decreases in the vein under pressure, it increases in the companion artery, both in breadth and brilliancy, until, indeed, we so retard the circulation that the entire vessel fades from view. The reason of this is obviously because the column of blood in the vein is increased in density, so that sufficient light does not penetrate to the tissues below to be reflected back; or, in other words, the light is absorbed by the denser fluid, while in the artery, on the contrary, the quantity of the blood is so much reduced that the light penetrates it even more easily, both in entering and returning, than in the natural condition of the vessel. Moreover, in certain conditions of congestion of the vessels, to be further dwelt on presently, in which no external pressure has been applied, the light-streak is very much diminished, or even entirely wanting.

Again, if no light passed through the vessel, and the light-streak was simply due to reflection from the red blood-column, then it would remain unaffected by the condition of the tissues lying posterior to the vessel. But the truth is, the light-streak is affected by such conditions, as is shown by the fact, that in separation of the retina it ceases where the separation begins, the vessel showing in the detached portion a uniform color across its whole diameter. This must be due to the fact that the light, after passing through the vessel, is diffused through the underlying fluid, and is consequently not reflected back.

The views advanced above will be made clearer by a diagram.

Let the following figure represent an enlarged section of the retina, in which the line A represents, we will say, the external limitans and choroidal epithelium, which we will assume to be the principal reflecting surface* of the fundus; and let the circle B represent a transverse section of one of the larger vessels in front of A.

Fig. 1.



We will suppose the fundus to have been illuminated by the ophthalmoscope in the ordinary way, and that the rays represented in the diagram are all return rays. Now these rays, whatever direction they may assume after passing through the dioptric system of the eye, must *start* from the reflecting surface A, as *divergent* rays. Thus the rays leaving the illuminated point C will be divergent, and strike as such on the vessel B lying in front. The central or less divergent rays which fall upon the vertex of the curvature of the vessel and its immediate neighborhood pass through the vessel and enter the eye of the observer; thus the centre of the

* We have assumed this for the sake of illustration. Light must, however, be reflected in a greater or less degree from all the different membranes at the bottom of the eye.

vessel appears illuminated; while, on the other hand, the outer or more divergent rays, striking on the descending portions of the vessels, do not in an equal degree enter the eye of the observer, and therefore the outer borders of the vessel appear darker, which may be represented *diagramatically* as the space included in the figure between the dotted lines EE, and the lines dd.

Thus we see that the effect, which Jaeger says is "the same as if produced by transmitted light," is, in fact, so produced; for if we look upon the reflecting surfaces of the fundus of the eye, after they have been illuminated by the ophthalmoscope, as the *source* of light, then we have the vessel between it and our own eye, and the conditions are precisely the same as those which pertain to transmitted light passing through a tube.

As this whole matter is merely a question of physics, the same phenomenon ought to follow wherever the conditions are the same as, or similar to, those which obtain in the eye.

Of course it is impossible to imitate artificially the conditions of the human eye, but a very simple experiment is sufficient to illustrate the principle upon which I think the effect produced depends.

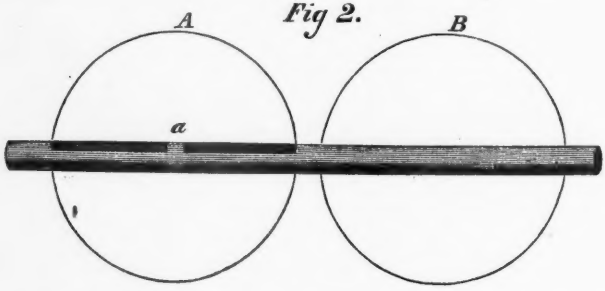
For this purpose I used two small cylindric boxes about three inches in length and one in diameter. These were fastened together longitudinally, like the tubes of an opera-glass. At the bottom of one of the cylinders I placed a mirror, made of London smoke-glass, silvered on the back. As the mirror was in fact an ophthalmoscopic

mirror, there was a hole in the centre, which fact I mention for a purpose to be explained a little later.

At the bottom of the other cylinder I placed a non-reflecting surface. A thin glass tube about a line in diameter, and containing a solution of water and carmine, was then run through the centre of both cylinders, just in front of, and nearly in contact with, the mirror in one and the non-reflecting surface in the other.

We may thus consider that one of these cylinders (Fig. 2, A) represents, in a rough way, the fundus of the

Fig 2.



eye, in which the mirror is the retina, and the red glass tube a retina vessel.

If now we cast the light, as in an ordinary ophthalmoscopic examination, down the cylinder containing the silvered mirror (which we will call A), so that the light from the instrument shall fall directly upon the glass tube, we get the appearance, along the centre of the tube, of a light-red streak, bordered both above and below by a dark-red line.

We see further, however, as we follow the light-streak

along, that as soon as we arrive at the central point of the tube, or that portion which lies directly over the hole in the mirror, the light-streak disappears and is replaced by a small quadrangular patch of a uniform color extending across the entire diameter of the tube.

If, however, we now look down the other cylinder (B,) we see that the tube, instead of presenting a light centre with darker red borders as in A, is uniformly colored throughout.

It is manifest that, if the light-streak seen in A was simply due to reflexion from the wall of the tube or from its contents, then we ought to get it also in B, as the tube is one and the same as that in A, and is seen, so far as it is itself concerned, under precisely the same conditions.

From this it follows that the difference in effect in the two tubes must be owing to some conditions external to the tube. Now the only difference in the conditions present is the difference in the nature of the two surfaces at the bottom of the cylinders.

It is thus evident that the light-streak in the centre of the tube in cylinder A, like that in the centre of the retinal vessel, is due to the same cause, namely, that the light passes through the tube and is reflected back from the mirror beyond.

That the reflexion does not take place from the posterior wall of the tube, or the posterior wall of the retinal vessel, but from the reflector beyond in the one case, and the tissues in the other, is proved from the fact that

where the glass tube passes over the hole in the mirror, that is, where the reflecting surface behind is wanting, the light-streak is also absent; see C, Fig. 2. This is evidently due to the fact that just where the hole is, the light, after having passed through the tube, does not strike upon the mirror, but passes through to the bottom of the box beyond, which being of a non-reflecting nature does not permit sufficient light to come back to the observer's eye to produce the light-streak.

To see precisely the same effect on a larger scale we have only to look down cylinder B, where the glass tube presents a uniform color throughout, for the reason just given above.

There are many interesting points in connection with this subject, both physiological and pathological, which I hope to discuss on some future occasion

ON ALTERATIONS IN THE HEARING OF ONE'S OWN VOICE.

By Dr. GUSTAV BRUNNER, of Zürich.

Translated from the German by J. H. Pooley, M.D., of Yonkers, N. Y.

Change in the sound of one's own voice is a not very uncommon accompaniment of ear disease, and forms sometimes a very troublesome symptom. It is found principally in diseased conditions of the middle ear (including the tubes), especially in acute or subacute cases, and generally appears on one side only, as these diseases are mostly one-sided. I do not here, of course, refer to those cases where, on account of the decrease of hearing or its total loss, the patients hear their own voice less distinctly or not at all, but only to those cases where the voice is heard of a different tone from the sound transmitted by the waves of the surrounding atmosphere. But even here different modifications are to be distinguished which have not yet been sufficiently examined, and which without doubt depend upon varying circumstances; although it seems to me that all have this more or less in common: that the individual's voice sounds dull, not as clear as formerly; that it has a hollow resonant

quality, and appears to have its seat within the head, while usually it is heard as if coming from without.

In other cases we find it sometimes weaker and duller, sometimes the person speaking has lost all perception of the loudness of his own voice, or it sounds disagreeably loud with a peculiar echo, and surpasses all other sounds, and becomes uncommonly unpleasant: it is of these latter cases that we intend especially to speak. Ear physicians have not until now paid particular attention to this subject, although the cause of this phenomenon is also interesting in regard to the physiological hearing of one's own voice. Gruber* has published, under the title of "Autophony and Tympanophony," a short communication on two cases of severe tubal catarrh, in which the patient's own voice as well as the respiration was not only very annoying to the patient himself, but was also strikingly perceptible by means of the otoscope, and only on one side. In the explanation of these phenomena, which he calls exaggerated autophony, or, if it is objectively perceptible by the otoscope, tympanophony, Gruber seems to me to be in error. Before I enter further upon this I shall communicate an analogous case from my own practice, which will, I believe, throw light upon this subject.

Madam Schenkel, 45 years old, had become deaf after having catarrh the whole winter, but had never before had any trouble with the ear; in April, 1870, sudden

* Monatsschrift für Ohrenheilkunde, 1868, p. 123.

pain in the right ear, without any distinct history of taking cold.

The pain was first felt upon blowing the nose, and was accompanied by sleeplessness, some dizziness (but without vomiting), considerable hardness of hearing, and continual roaring in the affected ear. No discharge from the ear.

When the patient called on me (about April 14th), I found that my watch was heard on the left side at a distance of 80 centimetres (instead of 150), and on the right side not even on contact with the auricle. Patient considered the left ear as sound, and says she has never suffered in her hearing.

She now does not complain of any particular pain, but of a feeling of stoppage of the right ear; particularly, however, that her own voice resounds very strongly in the right ear, in such a manner that speaking is very disagreeable to her; even her own respirations sound so loud and sibilant as to be troublesome; she also hears every motion of the jaws with unusual distinctness.

As long as she closes the right ear with her finger the loud sound of the voice ceases or becomes weaker. The examination showed the right membrana tympani free from perforation, somewhat dull, and in the upper part slightly reddened; in consequence of augmented concavity the malleus distinctly visible; not the slightest evidence of any accumulation in the middle ear. By auscultation with the otoscope one perceives quite a striking difference between the right and left ear. On the right

side the voice of the patient sounds uncommonly loud and hollow, with a tremulous continuation of tone; speech, even in common conversation, as though one was speaking in a vaulted chamber, or in a long speaking tube, and on raising the voice the sound is reduplicated; in quiet breathing, sound through the otoscope remarkably loud and sibilant (the beating of the heart could not be perceived, nor did the patient hear any sound from it). Of all these symptoms there was no trace on the left side: the auscultation here did not show the slightest abnormality. All these phenomena, subjective and objective, completely disappeared when the right ear was filled with water—the patient's own voice then sounded as usual. This was also the case upon stopping the auditory canal by strongly compressing the tragus; but upon opening the auditory canal all the symptoms reappeared immediately. I introduced the catheter on the right side, and blew air in by means of the air-bag, when I heard a strong roaring or blowing upon auscultation, which made, in spite of its intensity, the impression of a distant noise arising from the palate, or at most from the mouth of the tubes.

The air-douche was without permanent influence upon the phenomena. I now made, in view of Gruber's assertions—although from the result of the examination of the membrana tympani the presence of liquid in the tympanic cavity was not at all probable—a small perforation into the right membrana tympani.

The autophony was not altered by this proceeding.

and no air issued from the opening, in spite of strong and repeated douches of air through the catheter (the tube was no doubt still closed); as soon, however, as the air, after repeated efforts, passed through the opening in the membrana tympani with an audible hissing sound, the troublesome resonance, both subjective and objective, disappeared as if by magic, and did not return when I injected a weak solution of zinc by means of the catheter.

I remarked particularly that not the slightest secretion made its appearance through the membrana tympani. Now, the air passed whizzing through with great ease by the patient simply blowing the nose, and the improvement continued till next morning; in the evening, however, the patient was no longer able to force any air through the membrana tympani.

When I saw her two days afterward the opening was closed; no reaction had followed the paracentesis, but the group of troublesome symptoms were present as strongly as before. State of membrana tympani as in the beginning.

A tuning-fork placed on the temple of the patient was only heard with the right ear, and disagreeably loud and hollow; by introducing the otoscope during this experiment alternately into the right and left ear no difference was perceptible; this time the air-douche availed nothing, only after I had blown in some solution of zinc the resonance of the voice suddenly disappeared, and remained absent till the next morning. (To me this ob-

servation, which I subsequently repeated, is explained as follows: that after the moistening of the collapsed walls of the tubes the stream of air more easily finds admittance.) Two days later the patient presented herself again; the autophony continued with unabated violence; repetition of the air-douche was of no use, and even the injection of fluids at this time proved negative.

The patient stated that the air began to pass into the ears, but was suddenly interrupted. I now pushed a bougie of about 1 mm. in thickness through the catheter; at about the middle of the tube it was arrested, and could not be carried any farther, but the autophony had now disappeared; if I pulled the bougie back a little the symptoms reappeared, and disappeared again when the bougie was introduced. Thereupon I took quite a thin bougie, $\frac{3}{4}$ mm., which was also arrested at the spot mentioned, and found great resistance, but at last entered the cavitas tympani, whereupon the autophony ceased, even with the otoscope. I left the bougie in about ten minutes; the tuning-fork placed upon the temple was now, as formerly, only heard in the right ear, and disagreeably loud. The hearing-distance for the watch was not changed by the bougie; the patient, however, felt much relieved, and the continued roaring had somewhat diminished, but reappeared when the bougie was withdrawn. The autophony did not return, but the patient said before her departure that she felt as if the resonance of the voice would soon reappear, and so it happened. Regarding the further progress of the case I will be brief:

it proved very obstinate, and the course of the disease was on the whole similar to a chronic catarrh of the tubes and tympanum.

The autophony disappeared very slowly; but the intervals of freedom gradually became longer, and the troublesome symptom when it appeared disappeared again spontaneously. At first the catheter was always necessary to remove the autophony. Sometimes a single injection of air was sufficient, but sometimes it happened that it disappeared with the first pressure of the air, but returned with the second, and only disappeared completely after repeated injections.

I usually injected a solution of zinci sulph. (grs. iij.–ʒ j.), and I remarked particularly that the injected liquid never produced autophony, on the contrary, it disappeared, as I observed above, sometimes not from the effect of the air-douche, but by blowing in a few drops of the liquid. The injection very seldom caused burning in the ear, not even when I afterward several times injected a solution of nitrate of silver, grs. iij.–vj. ad ʒ j., which shows, according to my experience, that the liquid did not reach the cavity of the drum. The injection through the nose, according to the method of Gruber, which I now and then used in order to excite the fauces and mouth of the tubes (with sul. zinc. gr. iss ad ʒ j.), never succeeded; the sensation in the ear was nil, or quite inconsiderable, and the autophony did not give way (manifestly the treatment in this case was not sufficient to overcome the obstruction of the tube); the

amelioration after the air-douche usually lasted until evening, but sometimes till the following day; in the first case it was better in the morning upon waking (quite the same as in catarrh of the tubes), until, usually, upon blowing the nose the autophony reappeared. The patient says she feels then as if some thin obstruction, like a cloud, passed before the ear.

Violent sneezing would sometimes bring on autophony, as also merely stooping, whilst continued gargling often caused it to disappear. When the autophony is absent the patient feels much more comfortable, and says she hears better, although with the watch no marked difference can be noticed. The treatment consisted mainly of injections of solution of zinc (now and then also of sol. arg. nitr.) through the catheter, from 2 to 3 times a week; and more rarely Gruber's method was used. At the beginning I twice ordered four leeches to be applied to the mastoid process. Besides, the patient gargled constantly with alum, and snuffed up a solution of argent. nitr. (grs. iij - ʒ j); later I ordered also several nasal douches (mostly with a weak solution of salt and carb. potassæ). Patient is still (August 4th) under treatment.

The hearing distance, with the watch, increased by degrees from 0 to 16 centimetres, and she hears conversation much better. Valsalva's experiment gives no result. By means of the catheter air gains admission to the ear, not in a full stream, but only in jets, that is, one hears a sharp, abrupt, more or less moist crackling, without

roaring or blowing between. (Manifestly the air has difficulty in gaining entrance, a short jet passes, then the passage is closed again till the pressure is strong enough to overcome the resistance, and so on.)

The injected liquid seems not yet to pass into the cavity of the tympanum; it very seldom occasions burning, and then only to a slight extent. The injection of the membrana tympani has disappeared, and it shows instead a grayish cloudiness and some increase of concavity,—nothing particular. The continual roaring has not yet disappeared; it may be momentarily abolished by pulling the tragus from behind forwards. The autophony at present appears very seldom, and is never of long duration. Lately an unpleasant sensibility to some sounds produced outside of the body has made its appearance, which, according to the assertion of the patient, was not present to the same degree in the beginning of the disease. These are partly such as may be in part conducted through the body, such as the sounding of footsteps upon the floor, the opening and shutting of doors, the rattling of carriages, etc.; but likewise other sounds conducted through the air, as, for instance, loud music (especially certain tones), loud speaking or calling make a disagreeable impression upon the patient, and affect her head. The tuning-fork of C, placed upon the temple, occasions no disagreeable sensation in the right ear of the patient; but on holding it free in the air immediately before the right ear, then the sound was more unbearable than with the left ear.

POSTSCRIPT.

In the course of August the disease improved considerably, as well in regard to the hearing-distance as to the ceasing of the autophony; a relapse gave me an opportunity of examining the correctness of the above-described experiments. In regard to auscultation, I was able to convince myself fully that, during the continuance of autophony, the sound of the air-douche was strongly resonant, but always distant, as though arising in the fauces; when it approached nearer autophony had disappeared. The bougie had again the same effect, likewise the filling of the auditory canal with water, and I convinced myself by means of the otoscope that, in this latter experiment, the resonance had also disappeared *objectively.*

In making some critical remarks upon this case, I am well aware that the explanation of such difficult questions in physics presents many difficulties to the practical physician. I consider it, however, as our duty to labor in this common field according to our several ability. The history just related has strengthened me in my former opinion, that the phenomena described are phenomena of resonance, a vibration of the air in the middle ear, shut in by the closure of the tubes. (I shall call this symptom *tympanophony.*)

That the phenomena completely disappeared and reappeared by mere blowing of the nose, sneezing, or swal-

lowing, proves conclusively that they depend on very changeable conditions, and for this reason the opinion of Gruber, who considers them as *consonance-phenomena*, caused by swelling of the mucous membrane of the middle ear, is untenable.

We should be more apt to conjecture the presence of fluid in the drum, and its gradual change of position. In so far as tough mucus, or masses of pus, are capable of stopping up the tubes and thus producing in the drum, or perhaps also in the mastoid process, a closed air space, they might contribute, according to my view, to the development of resonance.

I believe that I convinced myself definitely that in this case there was no liquid collected in the cavity of the drum; on the other hand, I have seen many cases of accumulation in the drum, where the level of the liquid was distinctly discernible through the membrana tympani, and its presence confirmed by paracentesis where there was no tympanophony. Nor have I observed the phenomenon in question when adopting the practice of Gruber of injections through the nose, by which a considerable quantity of liquid finds its way into the cavitas tympani. It appears to me most natural at present to seek the cause of its frequent alternations in a stoppage of the tubes.

That such really existed is shown by the result of the auscultation, and by the fact that the solution of zinc injected through the catheter never or very seldom occasioned any burning in the ear. We may easily imagine that by means of the air-douche, if it is powerful enough,

the lumen of the tube is increased for a longer or shorter time, and mucus is displaced which had filled up the calibre or caused the walls to adhere. That, on the contrary, mere blowing of the nose or sneezing had a contrary effect is not surprising; it probably forced the mucus together so as to form a plug, without having the power to displace it.

For my view it may be further adduced that continued gargling sometimes abolished the troublesome symptom; that it sometimes disappeared spontaneously for a while, and that with its reappearance the patient had the sensation of something suddenly coming before the ear, or as if it became at once heavy and dull—complaints which are often heard in cases of obstruction of the tubes.

It might seem strange that tympanophony did not appear during the introduction of the bougie, but we shall cease to wonder at this when we consider that the tube is not a round but a fissure-shaped canal; the bougie cannot, therefore, fill up the whole cavity, but will act as a wedge holding the walls asunder. That tympanophony was not at once removed by paracentesis of the membrana tympani I explain by the small size of the opening, the edges of which easily reunited. I am well aware that I stand upon contested ground in my assumption, and that in the decision of the question of the normal opening and closing of the tubes (*i.e.* of the space under the styloid process), for which there are arguments pro and con., my explanation must also stand or fall. I am not at this moment in a condition to decide the

question theoretically whether or not the open condition of the tube is necessary to prevent resonance in the middle ear; however, it seems to me that the opening or closure of the Eustachian canal cannot be unimportant for the function of the ear. I cannot deny that tympanophony so strongly pronounced is very rare, while we frequently diagnosticate obstruction to the tubes. Whether in every case a closure of the space below the styloid process, or only of the remaining fissure of the tube, takes place, must be left undecided; besides I find, since I have paid attention to it, changes of one's own voice pretty frequently accompany catarrh of the middle ear.

Before proceeding any further, I must briefly touch upon the question as to the manner in which one's own voice is heard.

It is clear that the vibrations caused by speaking may reach our ears in different ways:—

1st. They will communicate themselves to the surrounding air, and then, like any other sound coming from without, will strike upon the membrana tympani.

2d. They will also propagate themselves through the solid structures of the body to the labyrinth. In this so-called bone conduction not only the vibrations are concerned, which, passing by the outer and middle ear, come directly to the labyrinth, but just as much those which proceed from the walls of the auditory passage (Helmholz *); and I add also those which pass from the walls of the tympanum to the air in the auditory passage

* Archiv für Ohrenheilkunde, vol. 5, p. 90, etc.

and the tympanum. This subject is not yet thoroughly understood.

3d. Lastly, it is possible that sound-waves proceed from the fauces, through the column of air in the tubes, provided these are open, into the cavity of the drum.

The last supposition is certainly the least worthy of consideration, as is plain from the following facts: it is shown by the researches of Voltolini,* and the modified experiment of Rinné, that the tone of one's own voice would necessarily be changed by every motion of swallowing, in which the mouth of the tube is opened; farther, if this were so, upon stoppage of the tube the voice should sound very weak, which is not the case.

It may be assumed that we hear our own voice principally in the first of these three described ways; not as if *none* of the vibrations conducted through the bones reached the labyrinth, but they will in comparison with the others produce only a weak sensation of sound, as our ear is formed for the reception of sound-waves through the air;† this is proved, among other things, by the experiment of Rinné, which A. Fick mentions in the "Anatomy and Physiology of the Organs of Sense," p. 141. If we place a vibrating tuning-fork to the teeth (or the bones of the skull), and permit it to sound out till nothing can be heard, we shall hear it again immediately if we place it close to the ear. Exactly the same

* Monatsschrift für Ohrenheilkunde, 1870, p. 39.

† When Lucae ("Untersuchungen über die Knochenleitung. Archiv für Ohrenheilkunde, vol. 1, p. 304") says, "above all, it is one's own voice which is specially conveyed to the ear by bone conduction," I cannot agree with him.

is the case (with regard to conduction through the tubes) with a tuning-fork permitted to sound out in the cavity of the mouth. If we further obstruct the conduction of our own voice through the outside air, which is best done by filling the auditory passages with water, we notice the following facts:—

1st. The voice sounds strikingly weak and distant.

2d. Even by loud calling it sounds not much louder than by low speaking or whispering, which is therefore comparatively more distinctly heard.*

3d. The voice seems to be seated in the middle of the head, whilst notwithstanding it is more external; this is very disagreeable to the person speaking.

By filling only one ear with water, the voice seems to sound principally in this ear; it sounds neither so weak nor distant as when both ears are filled with water, because the air conduction is still continued in one ear. The tuning-fork placed upon the head is only heard in the ear filled with water. Similar phenomena are observed if we place the hands over the ears whilst speaking.

* Schmidekam (Experimentelle Studien) found that when he dived with his ears full of water that he lost all perception of the loudness of his own voice. Those around were startled at the loudness of his voice while he thought he was speaking in his usual tone. This loss of perception related only to loud speaking, while speaking in a very low voice was perceived pretty nearly unchanged. This is in conformity with the results I have mentioned, only I must remark that in my experiments the person speaking had not entirely lost the measure of the loudness of the voice. This, however, will not seem strange if we remember that the experiments are not identical. Schmidekam spoke under water, by which the possibility of conduction of his voice through the air was more thoroughly excluded.

If this is done lightly, and without pressure on the auricle, the voice does not seem any weaker, but peculiarly resonant, tremulous, and in this way it may appear rather louder. By pressing the fingers tightly into the ears it becomes weaker; this, according to Helmholz, proceeds from the pressure on the root of the cartilage of the ear, for, according to this investigator, the so-called bone-conduction passes mainly through the cartilaginous portion of the auditory passage, and from thence to the contained air, and thence to the membrana tympani;* therefore the vibrations are diminished by this pressure. But these experiments are not quite decisive for our purpose, as the resonance of the air in the external meatus has some influence upon the result. That by filling the ears with water the sound-waves of the air are eliminated to a great extent cannot be doubted, and if we could be sure that in this way the bone conduction was not changed or weakened, the diminished sound of one's own voice might be referred solely to the absence of atmospheric vibrations. Lucae asserts in explanation of the louder sound of the tuning-fork in the ear filled with water,† that the bone-conduction is not augmented by the water in the ear, whilst the vibrations of the osseous canal are more easily transferred to the whole surface of the membrane, by means of water as a medium, than if the auditory passage was filled with air. Has Lucae not in this overlooked the consideration, that by the column of water

* Compare Lucae, Archiv für Ohrenheilkunde, vol. 5, p. 90.

† Ibid., p. 94.

resting upon the tympanum its vibratory power is diminished? Rinné's experiment seems distinctly to prove that our own voice is principally heard through the outer air, and I might add, that as soon as the air conduction ceases the voice is not only weaker, but also materially changed. The question may be asked whether, when we mostly hear by bone-conduction, the total impression will not be different from common hearing through the air, the resonance greater, and the sound somewhat hollow and tremulous in character. In order to decide this, it would be necessary, first, to examine whether the resonance be not rather a consequence of the methods employed in order to eliminate the ordinary conduction through the air.

Upon these relations rest some unexplained phenomena; as, for instance, it occurs to me that many hard-hearing persons use the English ear-trumpets (by Weiss of London) with striking advantage, whilst with others they do not make the tones any more distinct, but only give them a hollow, tremulous vibration; and it cannot be foretold in advance to whom they will be useful. At first I thought the effect ought to be proportioned to the size of the opening, *i. e.*, to the multitude of vibrations received; this, however, is frequently not the case; a lesser number may answer just as well as a larger.

I imagine that the so-called bone-conduction plays a part in it, and that the construction of ear-trumpets, and their application in individual cases, must be judged of from this stand-point.

Even the observation, that some patients in a car, where the noises are indirectly conducted through the body, will hear very badly, others, on the contrary, very well, has surely some relation to these facts.

To return to the history of our patient, the reason why only her own voice and breathing, but not the sounds produced outside and in the air, showed a strikingly louder and changed tone, can only be that, in the first instance, the source of sound lies in our own body, and that, beside the conduction through the outer air, the conduction through the solid parts exerts an influence.

From this circumstance we must explain the augmented resonance of the voice. Taking it for granted that complete closure of the tubes is favorable to resonance in the drum, it will appear nevertheless strange that resonance appears only in the sound of our own voice, and not likewise in that of others, and we are obliged to seek the conditions for this resonance in the conduction of the bones. One might, for instance, imagine that in this way, through the walls of the middle ear, or the external meatus, vibrations of the enclosed air would be more easily produced. It may appear strange at first, that by filling the meatus with water, or firmly pressing the tragus, the vocal resonance should cease, but we must remember that only one ear was diseased. If now by this means in the diseased ear not only the sound-waves from outside were shut off, but also the most of the bone-conduction was diminished

or paralyzed, the totally weakened conduction must reduce the resonance to a minimum, and so the individual's voice especially only be perceived by the sound ear.

That, according to Helmholz, the bone-conduction takes place principally through the cartilaginous portion of the auditory canal, and passes from thence to the air in the auditory canal and tympanum,—that it therefore becomes much weakened by pressure on the cartilage, we have seen above.

The same seems to me to be the case in filling the auditory canal with water, in spite of the contrary assertions of Lucae (see above), inasmuch as by the weight of the column of water the vibratility of the membrana tympani will be diminished.* The experiments with the auditory canal filled with water do not contradict the observations made above; what strikes us here is much less the resonance than the great diminution especially of the loud voice, and the sound appearing to emanate from the head. To be sure, one's own voice has a peculiar sound, somewhat dull, but so also has the voice of others. At all events, to sum up briefly, it seems to me indisputable, that obstruction of the tubes changes one's own voice in a peculiar and disagreeable manner.

We may convince ourselves of this in any severe

* I might here ask the question, whether the striking observation, that, in filling the auditory canals with water, the low voice sounds comparatively much louder than the loud voice, has its cause in the pressure of the water on the drum, which would offer more resistance to strong vibration than to weak ones ?

attack of catarrh; at present I am treating a young man with subacute catarrh of the middle ear, without exudation into the cavity of the tympanum, but with obstruction of the tubes, who hears his own voice in the manner described; after having blown in air several times by means of the catheter, the abnormal phenomenon disappears for some time. Only very seldom is the tone of his own voice so strikingly augmented as in the case communicated above; the changed perception is mostly only subjective, and cannot be objectively confirmed by means of the otoscope, as in the case just recorded. But the dull, hollow, indistinct tone of the voice which seems to come from the interior of the head is always characteristic, and makes speaking difficult.

I explain this, as already remarked, by resonance in the cavity of the tympanum, as in the same way resonance in the auditory passage produces similar effects.

HYPEROSTOSIS OF THE CRANIUM AND PETROUS BONES. PECULIAR MALFORMATION OF THE APPARATUS OF THE MIDDLE EAR. ANCHYLOSIS OF BOTH STIRRUPS. OSSEOUS CLOSURE OF THE LEFT FENESTRA ROTUNDA.

By Dr. S. MOOS.

Translated from the German by A. H. Buck, M.D., of New York.

In vol. ii., page 190 of the Archiv für Ohrenheilkunde I have described two cases of hyperostosis of the petrous bone with anchylosis of both stirrups. In the first of these cases there existed at the same time a narrowing of the openings and canals of the petrous bones, a condition of things which could only be explained by the extensive hyperostosis of both these bones. The fenestræ rotundæ, the external meatus, the porus acusticus internus, the canal of the cochlea, etc.,—all of these were very much reduced in size by the great increase in the volume of the bone. In the second case, however, there was a periostitis of the wall of the labyrinth, and the co-existence of a hyperostosis of the cranium and petrous bones should be considered as simply accidental.

Although the case which I am now about to describe resembles in many respects the two preceding, yet in one particular it is unique, as I shall show farther on. The petrous bones are the gift of my colleague, Dr. Weydung, who states that they were taken from the body of a man 49 years of age, who died of pleuro-pneumonia in the Evangelical Charity Hospital of Heidelberg. From his youth he had suffered from epilepsy, a disease endemic in his family, and had been deaf for the same length of time; on the left side totally, on the right to such a degree that he could understand speech only in the immediate neighborhood of the ear, but could nevertheless dispense with a speaking-trumpet. In consequence of this deafness his education had been very limited, but he was good-natured, trustworthy, and always ready for any work about the house. With the exception of this last illness he had never been acutely sick; he had also never suffered from syphilitic or rheumatic affections.

I omit as unimportant the post-mortem appearances of the lungs.

The skull-cap thick and compact, the diploë developed only in a few places, and then but slightly. The sutures obliterated. The dura mater adherent at all points, and very much thickened. Pia mater very opaque, the convolutions widely separated, and the lateral ventricles very much distended with fluid. The ependyma everywhere very much thickened, even at the origin of the striæ acusticæ. The plexus markedly œdematous. The brain

substance strikingly tough. The walls of the arteries, at the base of the brain, opaque, yellowish in spots, and very much thickened. Besides these there were no material changes in the brain.

RIGHT PETROUS BONE.

The osseous external auditory canal very thick and compact. Membrana tympani somewhat hyperæmic in the neighborhood of the manubrium; bright spot absent. The manubrium moderately drawn in. The mucous membrane pale and very much thickened. The roof of the tympanum very compact. The distance from the middle of the manubrium to the promontory 7 mm. Malleo-incudal joint almost though not entirely immovable. The head of the hammer and the entire anvil enlarged. The diameter of the head of the hammer 3 mm.; the diameters of the body of the anvil respectively 5 and 3 mm. The malleo-incudal joint proportionately enlarged. The diameter of the handle of the hammer near its middle point 1½ mm. The diameter of the short process together with the handle 4 mm. The diameter of the long process of the anvil 2 mm. The diameters of the short process of the anvil 3 mm. where it leaves the body, and 2½ mm. where it articulates with the posterior wall of the tympanum. The diameter of the os pisiforme 2 mm. The head of the stirrup somewhat lengthened, and of the same thickness as the os pisiforme. The stapedio-incudal joint does not lie free in the tympanic cavity, as is usually the case, but leans up

against an hyperostotic portion of the posterior wall of the tympanum. This osseous hypertrophy is so bounded anteriorly and superiorly by the canalis facialis as to overhang and completely hide the stirrup from view, with the exception of its lengthened head. It could not be decided experimentally whether the stapedio-incudal joint were hindered or not in its motions by contact with this hyperostosis, inasmuch as the base of the stirrup was found to be firmly united to the margin of the fenestra ovalis. The mucous membrane of the tympanum is everywhere slightly thickened; over the osseous hypertrophy it is very decidedly thickened, especially at the inferior portion near the floor of the tympanum, which is much reduced in size. The promontory remarkably small. The entrance to the fenestra rotunda normal. The musculus tensor tympani well developed; under the microscope the cross markings of the fibres perfectly preserved. Owing to the peculiar relations of the stirrup to the above-mentioned hyperostosis, the musculus stapedius could not be examined without injury to the specimen. The cells of the mastoid process normally developed only in the anterior portion; posteriorly they are to a great extent obliterated. The diameter of the bony wall of the mastoid process, which is entirely without any diploë, measures at the minimum $8\frac{1}{2}$, at the maximum 12 mm. The Eustachian tube viable, its mucous membrane pale. The bony substance of the labyrinth very compact. The roof of the vestibule and the walls of the semicircular canals of extraordinary thickness. The

space within the vestibule very large. The only morphological change appreciable is the great abundance of otoliths. In order not to injure the specimen no microscopic examination was made of the cochlea, especially as on the left side, where the functional disturbance had been even greater, only a negative result had been obtained.

LEFT PETROUS BONE.

The osseous external meatus the same as on the right side. Membrana tympani also the same, except that here the hyperæmia is wanting, and there is no bright spot. The mucous membrane near the periphery of the membrana tympani very opaque. Eustachian tube, the roof of the tympanum, and the ossicles the same as on the right side, except that here the ossicles are all anchylosed. Diameter of the head of the hammer, 3 mm.; diameter of the handle and short process together, 4 mm.; diameter of the handle below the point of insertion of the tendon of the tensor tympani, $\frac{5}{4}$ mm.; diameters of the body of the anvil respectively 5 and 2 mm.; diameter of the short process of the anvil, measured at the same spot as on the right side, $3\frac{1}{2}$ mm.; diameter of its anchylosed articular end, 2 mm.; diameter of the long process of the anvil, at the point where it leaves the body, 2 mm. in one direction, $1\frac{1}{2}$ mm. in the other; diameter of the os pisiforme, $1\frac{1}{2}$ mm. The head of the stirrup is lengthened out into

a bony process 2½ mm. in length and 2 mm. in breadth, so that the end of the long process of the anvil, together with the os pisiforme and head of the stirrup, present the appearance of a columella. The transverse diameter of the stapedio-incudal joint, 1½m. Notwithstanding the great length of its head, the stirrup appears as if walled in by the canalis facialis, the hypertrophied promontory, and posterior wall of the tympanum. In the midst of this hyperostosis is a furrow in which the stapedio-incudal joint and prolonged head of the stirrup lie imbedded. Above and behind this furrow the surface of the hyperostosis is somewhat knobbed. The fenestra rotunda lies between one of these knobs and the hypertrophied promontory. The latter is a large hemispherical mass of bone, reaching almost to the membrana tympani. Its substance is very hard and compact, like ivory; its surface is smooth. The entrance to the fenestra rotunda is so overgrown by the promontory as to present the appearance of a mere slit. Some idea can be formed of the thickness of this promontory when I state that, cutting at right angles to the prolonged axis of the cochlea, I removed with the chisel somewhat more than 4 mm. of bone without reaching the cupola. The remaining portion of bone measured 3 mm. in thickness; in other words, the diameter of the entire wall forming the promontory measured between 6 and 7 mm. In working out the house of the cochlea, the cuts being made nearly parallel to the axis of the cochlea, I found I was still 1½ mm. distant from the cupola. After

removing with care the eburnified house of the cochlea, I exposed to view the lamina spiralis membranacea of the first and second coil. As mentioned already, nothing abnormal could be detected in it. The trunk of the auditory nerve was also found to be normal. The osseous semi-circular canals hypertrophied. Diameter of the osseous portion of the superior semicircular canal, 4 mm. The roof of the left vestibule, 8 mm. in thickness. The mucous membrane and periosteum of the tympanum thickened, less anteriorly than posteriorly, but most markedly so over the hyperostosis and hypertrophied promontory.

As to the genesis of this case the question arises: Was the trouble congenital or was it acquired, or was simply the predisposition inherited, which was afterwards to show itself during the development of the cranium and petrous bones? In support of the first view we can only adduce suppositions, but no proofs. It is certain that infants are not unfrequently born into the world with compact and remarkably thick skulls; but, to my knowledge, nothing certain is known concerning congenital enlargement of the ossicles, promontory, etc. We only know that the ossicles of the new-born infant do not differ much in size from those of the adult. In the present case we are certainly justified in assuming that after birth an increase took place in the bony mass of the skull, which far exceeded the normal limits. Perhaps it was due to a diffuse periostitis, extending over the entire skull-cap and petrous bones; hence the

appearances already described of hyperostosis in different parts. If we take into consideration the immense thickness (7 mm.) of the promontory on the left side, and its ivory-like consistency, we are almost driven to the conclusion that here the osseous material was deposited from without, in consequence of a periostitis occurring after birth. To the same cause also was very probably due the enlargement of the ossicles.

In relation to the disturbance of the function of hearing, we consider the thickening of the ependyma at the origin of both acoustic nerves as immaterial, otherwise the deafness would have been complete on both sides, while in fact it was so only on the left side. The anchylosis of the stirrup and the osseous closure of the fenestra rotunda are amply sufficient to explain the functional disturbance. In this respect the present case resembles exactly the case referred to above, and published some time ago by me. There similar changes had taken place at both windows of the labyrinth, and the post-mortem appearances were amply sufficient to account for the functional disturbances which existed during life. These great changes rendered it impossible on the left side for the motion, communicated by waves of sound to the labyrinthine fluid, to find any yielding point at the fenestra rotunda; on the right side, however, it was still possible. It is remarkable in the present case that, although the trouble had existed for so long a time, no secondary changes had taken place in the nervous apparatus even of the left ear. This is another reason why

the hyperostosis of the skull-cap and petrous bones should be considered as the result of a periostitis. At the same time the condition of things observed in the labyrinth militates against the view that where anchylosis of the ossicles, especially of the stirrup, has existed for a long time, the terminal apparatus of the acoustic nerve undergoes a gradual atrophy, or fatty or colloid degeneration, through lack of the proper specific irritation.

SYMMETRICAL FORMATION OF EXOSTOSES IN BOTH EXTERNAL AUDITORY CANALS OF MAN.

By Dr. S. MOOS.

Translated by Dr. E. Gruening, of New York.

One who examines a great number of ear patients will be surprised at the relatively frequent existence of osseous tumors in the external auditory canal. The aurist will often discover them incidentally in patients consulting him for other alterations of the organ of hearing. Gout, rheumatism, syphilis, and purulent inflammations of long duration have hitherto been regarded the essential causes of the development of osseous tumors in the external auditory canal. These tumors may occupy every portion of the bony canal, but are most frequently observed in its middle third, where they generally spring from the posterior, anterior, or superior, and very rarely the inferior walls. They may be found either solitary or multiple, and in the latter case ordinarily growing from different walls simultaneously, thus encroaching considerably upon the free space. An accurate functional examination of such cases coming under our observation on account of other disorders, will, after the cessation of

the latter, hardly ever reveal an impairment of hearing, though there be only left between the exostoses a narrow fissure, or an opening of the diameter of a pin's head.

From what has been said above, one may infer how much the ætiology of these osseous tumors is still shrouded in obscurity and uncertainty, and I consider myself, therefore, the more entitled to publish this paper, as I believe myself to be able to state something positive concerning the genesis of certain exostoses in the external auditory canal.

Up to this time three cases of symmetrical formation of exostoses in both external auditory canals have come under my observation. Since, in all these cases, the tumors occupied identical spots on both sides, we may assume that the same cause was in operation everywhere. The tumors developed invariably from the upper wall of the external auditory canal, close to the drumhead, and opposite its upper pouch, the so-called membrana flaccida Shrapnelli.

As a rule, there existed a rather broad connection between the upper wall of the auditory canal and the tumor, which presented the shape of a heart on playing-cards, a white and stalactitic appearance, and a smooth surface free from all secretions.

An examination with the probe removed all doubt as to the nature of the tumors.

The ages of the persons were respectively 25, 43, and 58 years: the individual aged 25 was a girl; the other two patients were men. None of them had ever

suffered with rheumatism, gout, syphilis, or otorrhœa. They had never, in former years, complained of any disorder of their hearing. They all had recent catarrhal affections for which they sought and found relief, and enjoyed good hearing ever since.

These tumors, in all probability, constitute simple hyperplastic growths consequent upon irritative processes at the time when the annulus tympanicus coalesces with the squamous portion of the temporal bone, the union between which generally takes place before the osseous canal has fully developed from the annulus tympanicus by the accretion of new bone-substance in an outward direction, and toward the petrous portion of the temporal bone. We may therefore expect to find these tumors likewise in young individuals. As symmetrical structures, these growths bear a certain analogy to the osseous projections observed on both sides of the so-called spinous pelvis at the point of junction of the three bones, the pubes, ilium, and ischium, which constitute the os innominatum, and are originally separated from one another by the interposition of a cartilaginous layer. (See Virchow, Krankhafte Geschwülste, II., pp. 16 and 17.)

The tumors described above may exist on one side only. Of such cases I have already observed a large series.

Under certain circumstances these growths may assume a practical significance. If an operative procedure, *e. g.*, the removal of granulations in the region of the membrana flaccida, be required, they may render the performance of the operation very difficult.

RARE ANOMALIES, BOTH CONGENITAL AND ACQUIRED, OCCURRING IN THE EARS OF A DEAF-MUTE.

By Prof. S. MOOS.

Translated by C. J. Blake, M.D., Boston.

I am indebted to the courtesy of our former surgeon, Dr. C. O. Weber, for the rare specimen here described, and to his former assistant, Dr. G. Walz, for the somewhat meagre notes of the history of the case, as obtained from the relatives. The patient succumbed to an attack of purulent meningitis, during the first week following the removal of a large tumor from the right upper maxillary. Unfortunately the petrous bones, as I received them, were in a somewhat mutilated condition. As regards the hearing of the patient, the following only could be learned:—

V. K., æt. 50, peasant, from W——. As a child, was sickly; in his fourth year suffered from otorrhœa from both ears; according to the account of the relatives, a piece of bone was extracted from a fistulous opening behind the left auricle; in his fifth year the patient was attacked with ulcerated sore throat, and is declared (!)

to have become dumb in consequence. At the time of his admission to the Hospital he was perfectly deaf, and capable of uttering inarticulate sounds only. At the inner end of the right external meatus was a large plug of cerumen. The left external meatus was almost entirely closed by a crescentic membranous fold, having a small opening in its lower portion. Behind the auricle, near the end of the inferior third, and in the furrow between it and the mastoid process, was a cavernous depression, at which point the piece of bone was said to have been extracted in childhood.

I will give the report of the results of the post-mortem, made by Prof. Jul. Arnold, only so far as they concern the cranial cavity. Crown of the skull thin, compact, diploë scanty. In the sinus longitudinalis a fresh fibrine clot; the inner wall irregular and rough, in consequence of numerous soft granulations projecting into the sinus. The outer surface of the dura mater, at the apex, markedly injected; the inner surface to the right and left covered with a purulent fibrinous deposit; the underlying structures swollen, dull, and injected in spots. In the space between the dura and pia mater were several ounces of a yellowish-green purulent fluid.

The pia mater, over the left hemisphere, at the point corresponding to the parietal lobe, showed decided vascular congestion, together with swelling and sponginess of the structure; it was, however, transparent, and only flecked with pus; along the sinus longitudinalis, on the contrary, it was very dull, thick, and velvety, and almost

entirely covered by purulent deposit. On the right side the pia mater, throughout its whole extent, was very dull, thick, and spongy; the upper surface irregular, slightly eroded in spots, and exhibiting great vascular congestion, and a purulent fibrinous deposit over the entire surface.

The dura mater, at the base, had undergone the same changes as at the apex, but in a lesser degree, though most decidedly on both sides of the clivus. In the sinus, at the base, there was dark fluid blood and clots, both fresh and firm. The pia mater, at the base, showed the same changes as that over the left parietal lobe. At the anterior end of the left squamous lobe, and resting upon the pia mater, was a soft, vascular body, of the size of a bean, the greater portion projected above, the lesser portion being situated within the pia mater and in the substance of the brain itself. It was grayish-red in color, very anæmic, the interior portion broken down, the centre showing merely the remains of a network figure. The dura mater, at the point corresponding to the tumor, was defective, as was also the inner wall of the base of the cranium, almost immediately below the sharp posterior boundary of the cavity.

A considerable portion of the outer wall of the orbit posteriorly was defective, so that there were two large openings communicating with each other: of these the smaller was below; the upper and larger opening led into the orbital cavity, and was closed merely by the periorbital membrane. In the former opening there was a red-

dish purulent fluid. The ventricles were pretty large, containing an almost clear fluid. The substance of the brain contained a moderate amount of blood, and was of medium consistence. In the cerebrum there was nothing remarkable.

The substance of the cerebellum and that of the pons was normal; the ependyma normal. The striæ acusticæ were clearly marked. A further examination showed the deeper roots of both acustici perfectly formed and well developed. The growth mentioned was found by subsequent microscopic examination to be a glioma.

RIGHT PETROUS BONE.

The cartilaginous portion of the external meatus as well as its dermoid covering showed no marked anomaly. The inner end of the meatus was perfectly closed by a tough ball of cerumen, on removing which the part beneath appeared of a bluish-red color; this included the region of the membrana tympani, which was entirely wanting. The meatus terminated, so to speak, in a blind spherical cavity, lined with its periosteum, and of somewhat larger dimensions than the usual diameter of the inner end of the meatus in the adult. On removing the dermoid covering an osseous mass was everywhere found, even in the place of the membrana tympani. The styloid process was very well developed.

Middle Ear.—In the specimen, as I received it, the cartilaginous portion of the Eustachian tube had been

cut away close to the bone, so that unfortunately I could not ascertain its condition. The tegmen tympani was very thick and compact; the mastoid cells but moderately developed. The greater portion of the middle ear had undergone osseous obliteration. The passage of the osseous portion of the Eustachian tube was represented by an opening the size of the point of a pin (from a transverse section made at this part of the preparation); this opening was surrounded by a firm, ivory-like bony mass. The canal or semi-canal pro tensore tympani was obliterated, the musculus tensor tympani, malleus, and incus were wanting. On chiselling away a part of the bony structure, there was still further evidence of the fact that the membrana tympani had been replaced by a mass of bone. The tympanum, including the passage to the fenestra rotunda, as well as the fenestra itself, had undergone osseous obliteration, with the exception of a small cavity near and below the stapes, into which the head of a pin could be pushed downwards and backwards to a depth of $3\frac{1}{2}$ mm. This was rendered easier as the posterior crus of the stapes was wanting, being represented by an obtuse bony protuberance; the posterior portion of the base of the stapes was also wanting. The remainder of the stapes, that is, the head, somewhat elongated; the anterior crus and the anterior portion of the base were immovably united, the former to the osseous mass in the tympanum, the latter to the bony rim around the fenestra ovalis. In consequence of the defect in the base of the stapes there existed a correspond-

ing opening in the wall of the labyrinth, covered only by periosteum.

The canalis facialis, together with the nerve, was normal. The space within the vestibule was diminished by two smooth conical exostoses running across it.

The upper and larger was 3 mm. long and about 2 mm. thick, and passed obliquely from that part of the lateral wall of the vestibule which lies nearly above the fenestra ovalis, to terminate just below the anterior ampullar opening of the horizontal semicircular canal. The lower exostosis was 1 mm. long, barely ½ mm. thick, and extended from the posterior end of the incomplete base of the stapes to the point of greatest convexity on the median wall of the vestibule. The entire distance from the vestibular surface of the stapes to the median wall was not greater than the length of this small exotosis, namely, 1 mm. The unusual convexity of that portion of the median wall of the vestibule towards the floor did much to diminish the middle diameter. The transverse diameter in the upper part of the vestibule was 4 mm., the antero-posterior 4½ mm., the decrease in space affecting particularly the inferior and posterior portions. The walls of the membranous sacculi, which contained a remarkable number of otoliths, were very thick. The osseous and membranous semicircular canals, the cochlea, and the osseous and membranous spiral laminæ were fully developed, the one remarkable point being the general sclerotic appearance of the osseous cochlea and the partition walls of the various whorls.

LEFT PETROUS BONE.

Outer and middle ear.

Upon enlarging the small opening already described, there was found a collection of numerous scale-like masses, of varying size, resembling epidermis, having a greasy lustre and overlying one another like the coats of an onion. After its removal the absence of all the essential anatomical attributes of the outer and middle ears was evident. There remained inferiorly the styloid process, and anteriorly the osseous boundary of the posterior wall of the glenoid cavity.

In the middle ear there remained only the cartilaginous portion of the Eustachian tube and the apophysis mastoidea. The lower end of the canalis facialis, with exception of the inner wall, just before its exit from the foramen stylo-mastoideum, was denuded (usurirt) for the distance of about 1 cm. (there had been no signs of paralysis affecting the left facial during life). The internal muscles of the ear and the osseous Eustachian tube were wanting. In their place there was only a large cavity filled with the mass above described, extending forwards as far as the canalis caroticus, and backwards to the sinus lateralis, with both of which the outer surface of the mass was in close contact, while a part of it had forced its way through the base of the petrous bone as far as the inner end of the porus acusticus internus. The so-called labyrinth wall, or inner wall of the tym-

panum, was found, after removal of the mass, to be concave instead of convex.

Above and behind this concave space were a few bony growths, irregular, rough, knotty, and as hard as ivory. All the parts belonging to the labyrinth wall, the fenestræ, promontorium, canalis semicircularis horizontalis, stapes, etc., were wanting. The whole of the internal ear had undergone osseous obliteration; as a result of this, the vestibule, cochlea, and semicircular canals were wanting, only the porus acusticus internus and the auditory and facial nerves remaining intact. Unfortunately, these latter were injured in preparing the inner ear, so that I cannot speak of the microscopic appearances of the terminations of the nervus acusticus. The microscopical examination of the tumor showed it to possess all the characteristics of a cholesteatoma, or the so-called "pearly tumor." The overlying layers, like the coats of an onion, were found to consist largely of fatty epithelial cells, between which lay numerous rhombic crystals of cholesterine. In many places, especially towards the centre of the cavity, the fatty degenerate masses formed genuine atheromatous conglomerations. I could determine neither calcareous degeneration of the epidermic cells, nor even a calcareous appearance, nor the chalky metamorphosis in the central portion of the tumor mentioned by Virchow (Die krankhaften Geschwülste, Bd. 1, S. 229) as characteristic of atheromata which have ceased to develop. With regard to the origin of the tumor I shall speak further on.

On a closer consideration of the anomalies above described, it is clear that we have to do with congenital as well as with acquired changes.

I.

CONGENITAL ANOMALIES.

A. In the Right Petrous Bone.

The three divisions of the ear are developed independently and in separate parts. The external ear (membrana tympani, meatus, and auricle) are developed by metamorphosis from the first embryonic branchial cleft, of which, in the middle ear, the Eustachian tube is the remnant (Bischoff, Kölliker, Valentin). From the first branchial lappet, according to Reichardt, are formed the malleus and incus, and from the second branchial lappet, the stapes, styloid process, and the short horn of the hyoid. In our case, therefore, there had been an arrest of development in the first branchial lappet especially, as the membrana tympani, malleus, and incus were wanting, and the Eustachian tube was obliterated, while the styloid process and the stapes were present, the latter however, imperfect. The exostoses in the vestibule are to be considered as accidental accompaniments of the irritative processes in the middle ear at the time of ossification, and by no means as independent irregularities of development, especially as one of the exostoses originated from the base of the stapes, close to the point

where the vestibule was separated from the tympanum by periosteum only, and the other near the same spot; after birth in this case both tympanum and vestibule were, in all probability, nourished from the same source.

This case may rather be taken as a further proof—

1. That during the various and independent developments of the several sections of the auditory passage, derangements or interruptions may occur in the development of one part while the other remains unaffected.
2. That the middle ear and the labyrinth develop independently of one another. The contrary view was formerly taken by Hyrtl (Beiträge zur Entwickelungsgeschichte des Gehörorgans: Oesterreich. med. Jahrbücher, Bd. xx., S. 449).

According to Hyrtl, tympanum and labyrinth are formed from a common sinus, by means of its division into an inner and outer portion.

The period of the arrest of development in our case was probably within the third and fourth months of embryonic life.

B. In the Left Petrous Bone.

The whole of the internal ear was obliterated. The semicircular canals, vestibule, cochlea, and both fenestræ were wanting. Montain and Saissy (compare Lincke: Handbuch der Ohrenheilkunde, vol. I., p. 630, et seq., and Moos: Klinik der Ohrenkrankheiten, pp. 341–48) observed the absence of the vestibule, cochlea, semicircular canals, and both fenestræ in deaf-mutes. All of these

conditions can be considered only as the result of arrest of development at the earliest possible period—the first week—in the labyrinth vesicle. The existence of the auditory nerve accompanying absence of the labyrinth is nothing remarkable, as it is independently developed. The condition of the middle ear is more difficult of explanation. As above stated, only the cartilaginous portion of the Eustachian tube and the apophysis mastoidea remained, the membrana tympani, with the accompanying ossicula, were wanting. The presence of the cartilaginous portion of the Eustachian tube gives greater probability to the conclusion that there was not an arrest of development in the first branchial lappet. That there was an arrest of development in the second branchial lappet seems to me improbable, notwithstanding the absence of the stapes, particularly as the styloid process, which is developed from this lappet, existed. We cannot but consider the state of things in the middle ear as of congenital origin, or rather as a combination partly congenital and partly acquired.

II.

THE ACQUIRED ANOMALIES.

These are very instructive in relation to the changes exhibited in the petrous bone in connection with the cholesteatoma, and the case is furthermore characteristic of the origin of these tumors in the ear. The tumor is a secondary phenomenon. According to the history, the

patient had in childhood a purulent inflammation which resulted in necrosis. Atresia of the external meatus occurred probably at some time before the cessation of the purulent inflammation, and from the time of the closure of the meatus the epidermis collected under the same conditions as in the sebaceous tumors of the skin, which possess an actual sac. From the enormous size of the tumor, which extended from the external meatus to the sinus transversus, the canalis caroticus, and so far as the porus acusticus internus, the symptoms of irritation may have appeared a long time after the closure of the external meatus—how long after it is exceedingly difficult to determine. The defects existing in the osseous portion of the temporal bone may be considered principally as the results of the pressure of the steadily-increasing tumor, and to but a slight extent as due to the necrosis occurring during childhood.

In connection with the existing atrophy of certain parts, in consequence of the pressure of the tumor, it is an interesting fact that the cartilaginous portion of the Eustachian tube remained intact while the osseous portion was obliterated. The softer, and therefore more mobile structures can more readily give way before and evade pressure.

PRACTICAL INFERENCES IN REGARD TO TREATMENT.

Toynbee was the first to describe and to give a true estimate of the importance of cholesteatomata in the ear,

under the unfortunate term "molluscous tumors," for which he subsequently substituted the name "sebaceous tumors." (Compare Pathological Researches into the Diseases of the Ear, Seventh Series. Sebaceous Tumors in the External Meatus; their Effects upon the Organ of Hearing, the Petrous Bone and the Brain, with Suggestions as to their Treatment, by Jos. Toynbee. Medico-Chirurgical Transactions. Vol. XLV., and his work upon Diseases of the Ear: German translation, p. 118.) After an anatomo-pathological description, Toynbee proceeds to speak of the treatment, and among other points justly lays stress upon the following rule:—In the advanced stage, when the base of the tumor has buried itself in the petrous bone, the greatest care is necessary in opening it, as it may be in contact with the dura mater. Bearing in mind the condition of the left petrous bone, as above described, we feel it our duty to draw particular attention to the precaution advised by Toynbee, especially in view of a method of treatment for purulent inflammation of the ear, accompanied by perforation of the membrana tympani, which has of late become a favorite. For the purpose of thoroughly cleansing the middle ear from secretion, when there is a perforation of the membrana tympani, Hinton adjusts the nozzle of the ear-syringe, covered with rubber, in the meatus, so that the passage shall be, as nearly as is possible, hermetically closed, in order that the fluid which is injected—the Eustachian tube being sufficiently permeable—shall pass through, and flow out at the nose (James Hinton: Further Obser-

vations on Catarrh of the Tympanum, Guy's Hospital Rep. XV.). Lucae, referring to this paper in the Med. Centralblatt, No. 20, 1870, adds, that he had long since convinced himself of the feasibility of this method, but finds it still more expedient to follow the suggestion of Prat, and employ a double canula, with which the passage of the fluid through even the smallest perforation in the membrana tympani is attained with greater certainty and less danger of injury; the injection of fluid in this manner being at any rate much more rational than the injection of fluid (catheter) or air (Valsalva's and Politzer's methods) in the opposite direction, as the latter, in place of cleansing the tympanum, forces into it the secretion which is in the Eustachian tube.

For my part, I consider the old method as much the less dangerous; it may be employed without hesitation in every case, and this cannot be said of the newer procedure; neither is it less reasonable. In very many cases of purulent catarrh there is an indication for the opening of the Eustachian tube, which is easily accomplished by Politzer's air-douche, when repeated attempts, by means of continued injections through the meatus, prove futile. In many cases, where the opening in the membrana tympani is a small one, Politzer's method works admirably, the external meatus being filled with a medicated fluid at the time of the air-douche, the injection of the air is often followed, almost immediately, by a discharge through the Eustachian tube into the pharynx of the fluid which has penetrated into the tympanic cavity. With this method,

moreover, there is no danger of an injury to the vital structures within the cranium lying near the tympanum, even when there is a solution of continuity between the two cavities.

OBSERVATIONS ON CASES OF PARALYZED ACCOMMODATION.

By Dr. COLSMAN, of Barmen.

Translated by Ch. F. Rodenstein, M.D., Westchester, N. Y.

Accommodation, it is well known, consists in a demonstrable increase of the convexity of the anterior surface of the crystalline lens, produced by the action of the ciliary muscle. During childhood and youth, while the lens is still soft, it has the disposition to assume as convex a form as its attachment to the zonula Zinnii will allow. By the action of the ciliary muscle the zonula is relaxed, and the lens acquires the liberty to assume its full convexity. With the cessation of the muscular contraction the zonula is stretched (perhaps by the pressure of the vitreous from behind), and the lens is flattened. As long as the lens retains its pristine elasticity every muscular contraction will cause an increase in its curvature by relaxing the zonula. But when the lens becomes more rigid, even the strongest muscular contractions fail to heighten its convexity; simply because it no longer possesses a sufficient amount of elasticity to change its form essentially.

It is a remarkable fact, which at first seems to contradict all other observations made with regard to the growth of muscular power, that the range of accommodation is greater in a child of ten years than in persons of twenty or thirty; that in fact it decreases from year to year, while muscular strength develops with ripening manhood up to its very prime. This apparent contradiction finds its explanation in the circumstance that the lens, which in early life is soft and elastic, becomes harder and more rigid with advancing age, and less disposed to change its form. The limitation in the range of accommodation, which can be shown to exist even in early youth, depends therefore not upon a diminution of power in the ciliary muscle, but upon a decrease in the elasticity of the lens; although it is quite probable that, in advanced age, the decay of power in the tensor choroideæ may contribute, with the progressive rigidity of the lens, to produce presbyopia.

But, besides, in cases of ordinary presbyopia,—the limitation of accommodation dependent upon old age,—cases of paralyzed accommodation are not unfrequently observed in all ages, in which, for evident reasons, only a decrease in the muscular power of the tensor choroideæ can be assumed. Thus, for instance, we cannot ascribe to a change in the consistency of the lens that paralysis of accommodation which is dependent upon the instillation of atropine, or upon an affection of the nervus oculomotorius, from which the muscle of accommodation receives its enervation, or upon general debil-

ity, resulting from hemorrhage or other exhausting diseases. In such cases the lens plays but a passive and subordinate rôle. That the muscle of accommodation is here at fault may be easily perceived, as soon as it is recollected, on the one hand, that by its appropriate stimulant, the extract of calabar bean, an act of accommodation can be called forth, which, though transitory, is similar and equivalent to the normal; and on the other, that this disturbance of vision may pass away slowly, but completely, on the return of bodily strength in restitutio in integrum.

In many cases of paralysis of accommodation the sphincter of the iris is implicated in this interruption of mobility, especially when the cause of the disease has its seat in the nerve or the brain. The action of the iris is less frequently disturbed when paralysis occurs in consequence of fatigue or over-exertion of the ciliary muscle. It will be difficult to state a fixed rule concerning the coincident occurrence of these two affections.

Moderate diminution in the energy of the muscles of accommodation, such as accompanies general debility, or follows febrile affections, is evidently most likely to act as a disturbing cause in such cases, in which an unfavorable condition of the refracting media has already made large demands upon the powers of the ciliary muscle. This is particularly the case in hypermetropic eyes. Here the powers of accommodation have already been largely called into requisition to correct errors of refraction; and persons so afflicted are made uncomfortably

conscious by their inability to perceive small objects distinctly, or by a disagreeable sense of fatigue, that their powers of accommodation, which heretofore have been just sufficient, are no longer able to render them their necessary service.

But even in eyes of normal structure a weakness of accommodation, which is not the result of age, may produce asthenopic troubles, especially if it appear suddenly. This occurs still less frequently in persons afflicted with myopia of the higher grades, for they make generally but small demands upon their range of accommodation; and a diminution of it is scarcely perceived.

The *causes of paralysis in accommodation* are often the same as those which are apt to produce general muscular debility. Among these may be counted: first, defective nutrition of the muscular and nervous system, hemorrhages, febrile diseases, and in general all debilitating influences, which weaken also the apparatus of accommodation, consisting as it does in its active element of muscular tissue; then paralysis of the nervus oculomotorius, which presides over the function of accommodation; and finally, exhaustion of the muscle of accommodation by ascertained over-exertion—in which case, however, we are disposed to look for the seat of the affection, rather in the substance of the muscle than of the nerve. Wounds also, and blows and bruises, as they sometimes produce paralysis of the iris, may also produce a similar condition in the muscle of accommodation.

It appears that diseases of particular organs sustain a

specific causative relationship to the weakness of the muscle of accommodation, but which is not yet clearly understood. Thus many observers have shown that paralysis of accommodation is not a very rare concomitant of diphtheria. I have seen considerable weakness of accommodation even in a simple angina, in which the attending physician could see nothing of a diphtheritic inflammation. Dr. H. Schmidt called attention to a peculiar relationship subsisting between paralysis of accommodation and toothache, and showed, by comparative measurement, that among about a hundred persons, who suffered from toothache, a large number had paralysis of accommodation on the affected side.

Secondary syphilis deserves a prominent rank among the causes which—probably by affecting the central or peripheral portions of the nervus oculomotorius—produce paralysis of accommodation, as well as of the sphincter of the iris.

In the following pages a few of the most interesting cases of paralyzed accommodation and their probable cause are reported, as they were observed from time to time at my clinic.

CASE NO. 1.—*Sudden Paralysis of Accommodation after undue Exertion.*

An architect, aged 27 years, while engaged in drawing, noticed that all at once he could no longer see to read. On examination it was found that all the visible parts of the eye were sound; that the acuteness of vision for distant objects was good; and that no evident errors of refraction existed. Ordinary printing could not be read; but with

a convex No. 14, Jaeger's No. 1 could be read. There was evidently a sudden paralysis of accommodation of both eyes, produced by intense over-exertion of the muscles of accommodation.

In view of the age of the patient, and with the probability that with rest the ciliary muscles would soon recover their accustomed strength, a favorable prognosis was made.

The patient was advised to abstain from using his accommodation as much as possible, but to use, in case of necessity, convex glasses No. 14 for near objects, by which small print could be easily read.

The result of the prescribed means was that the young man could again read small print, as he wrote to me two months afterwards. The muscles of accommodation had therefore recovered a sufficient degree of activity.

Case No. 2.—*Paralysis of the Sphincter of the Iris, and of the Ciliary Muscle on one side, and beginning Paralysis of the Ciliary Muscle on the other, after straining the eye during work over a bright fire.*

A glass-blower, aged 36 years, healthy, complained of failing sight in the right eye for several months. The right pupil is considerably dilated; the left is normal. No pathological changes are visible. The acuteness of vision for distance is most improved by convex No. 24, with which an approach to the normal standard is obtained. There exists, therefore, hypermetropia $=\frac{1}{24}$. It requires exertion to read Jaeger No. 10 at 28 inches with convex No. 24; without a lens it is impossible. There is, therefore, paralysis of the ciliary muscle, with a range of accommodation only $=\frac{1}{28}$. With convex No. 8, Jaeger No. 1 is read. An application of a concentrated solution of calabar bean enabled him to read also Jaeger No. 3 within $\frac{1}{2}$ foot. The pupil contracted with precision after instillation of calabar bean, and by the use of this drug six times per diem remained contracted for several days.

The left eye was emmentropic, of unusual acuteness, but showed a considerable reduction in the range of accommodation. He read Jaeger No. 3 at 18 inches; No. 1 and 2 not at all. His accommodation at the best was $=\frac{1}{18}$, which at his age is certainly abnormal.

The origin of this affection may be attributed with greatest probability to the employment of the patient. According to his statement he had to spend ten hours every third day in blowing glass cylinders. His work required great attention. These cylinders were exposed to the heat of a bright coal-fire, and when red-hot he gave them their definite form with a blow-pipe. It is evident that the constant contemplation of such bright and glowing objects must call the activity of the apparatus of accommodation and the retina into considerable exercise. The permanent dilatation of the pupil was the necessary consequence of the constant gaze upon a bright fire; and it is not improbable that the constant stretch of the muscle resulted in an entire loss of its contractility. The examination of the patient furnished no other elements which might be used to construct an etiology for this affection.

Case III.—A lithographer, aged 38 years, complained, in 1867, that he was no longer able to pursue his avocation without great fatigue for any length of time. His work made, indeed, very large demands upon his accommodation. Latent hypermetropia was with reason suspected, and on examination it was found that the perception of smallest objects was facilitated by the use of convex No. 50.

In 1869 he reported that he had been able to get along very well in his work with the aid of his glasses; but that for three weeks he was very much troubled with asthenopia whenever he attempted to read or to work. Immediately before, he had suffered from an acute disease, with angina and rheumatic pains in the neck.

On examination it was found that the power of accommodation, which before had concealed existing hypermetropia, now was reduced to such a degree as to make the latter affection manifest. It amounted to $\frac{1}{40}$. This discovery explained the asthenopic troubles. The febrile affections and angina were certainly implicated in reducing the energy of accommodation, and furnished thereby the occasion for the weakness of vision. Whether the angina was of a diphtheritic nature or not, could not be ascertained.

Attention was paid to the weakness of accommodation by ordering

for him Convex No. 36. With $\frac{1}{36}$ he could neutralize his hypermetropia $\frac{1}{40}$, and have at his disposal the whole range of accommodation.

CASE IV.—*Paralysis of Accommodation as a sequel to Rubeola.*

Quite an intelligent little girl, of about 6 years of age, complained, after an attack of measles, that she could no longer read. The parents state that she could read very well before she was sick.

There were no pathological alterations. Jaeger No. 15 could not be read. No. 16 was still perceived. Hypermetropic structure could not be demonstrated. With a convex No. 8 Jaeger's No. 2 could be read. Hence the whole affection consisted in a deficient focal adjustment of the dioptric apparatus, inasmuch as no hypermetropia existed. The indications, therefore, were to raise the powers of accommodation, which, with the failing of the general tone of the system, had become depreciated. Invigorating means were ordered, nourishing diet, with Tokay wine, and under this treatment the power of accommodation increased $\frac{1}{24}$ in a week; inasmuch as Jaeger No. 10 could be read at 24 inches. In three weeks the range of accommodation was tripled. Jaeger's No. 2 could be read at 8 inches.

Two months afterwards every trace of paralysis had disappeared. No. 1 was read easily, and the little patient could again attend school. Later information confirms the complete recovery from weak sight.

This case of paralyzed accommodation is of interest, on account of the extreme diminution of the range of accommodation and the rapid recovery of lost power.

CASE V.—*Paralysis of Accommodation after Angina.*

A bright little girl, 6 years old, was all at once unable to decipher letters in reading. By testing vision it was found that Jaeger No. 14 could still be read, but not No. 13. No pathological alteration could be discovered in any portion of the eye. The ophthalmoscope, however, revealed a strong hypermetropia. The musc. rect. sup. of the left eye was slightly paralytic.

I learned that the child had lately suffered from sore throat. And as diphtheritic angina is apt to be accompanied by paralysis of the muscles of the eye, so this case of angina was attended, besides paralysis above referred to, by a loss of the power of accommodation. With pre-existing hypermetropia this was, of course, still more evident and troublesome. The child read Jaeger No. 4 with convex No. 8. Unfortunately the parents did not return, probably from pecuniary motives, as I had expressed myself assuringly quoad restitutionem.

Probably the trouble was very soon relieved.

CASE VI.—*Paralysis of Accommodation after Angina.*

A boy, aged 6 years, asserted that he could no longer distinguish the letters in his reader. He read, indeed, only Jaeger No. 15. Hypermetropia=$\frac{1}{18}$ was discovered. No other abnormities in any organ can be seen. Jaeger No. 3 can be read with convex No. 8. I have no doubt that the patient could have read Nos. 1 and 2 if his backwardness in reading had not made it difficult for him to recognize small letters. We have here evidently to deal with a considerable limitation of accommodation in a hypermetropic eye. The only question is as to the cause of this sudden paralysis. I ascertained that, several weeks before, the patient had suffered from angina, unattended by fever, but during which slight superficial plaques became apparent upon the tonsils. There was no reason for looking upon this disease as diphtheritic in character. But, notwithstanding, we are forced to assume a specific and causative relationship between this benign angina and that sudden paralysis of accommodation. No other sufficient explanation can be given for this sudden occurrence of loss of accommodation. Neither the general reduction of strength after a slight angina (the patient felt perfectly well), nor the assumption that this hypermetropic patient over-exerted his already deficient powers of accommodation, can be looked upon as adequate causes; for, in similar cases of insufficient accommodation with hypermetropia, asthenopic difficulties generally precede a permanent relaxation of the ciliary muscle.

The treatment in this case consisted in absolute rest from efforts of

accommodation; in the constant use of convex No. 20; in supporting diet, assisted by iron and Tokay.

CASE VII.—*Recent Paralysis of the M. Sphinct. Iridis and Musc. Ciliaris of one side without known cause.*

A tailor, aged 46 years, suffers for 8 days from mists before the left eye. Examination reveals, as the cause of this, moderate mydriasis, with weakness of accommodation. Small print cannot be read by the otherwise emmentropic eye. Extract of calabar bean produces sufficient contraction in the apparatus of accommodation to enable the patient to read Jaeger No. 3. The pupil also contracts. With No. 14 the finest test of Jaeger is read. After a free use of calabar bean Jaeger No. 5 is easily read at 14″ without glass and without the drug. This proves that the range of accommodation has become again nearly $= \frac{1}{4}$.

CASE VIII.—*Paralysis of Iris and Accommodation in Syphilis.*

A merchant, aged 30 years, has suffered for two months from a moderate dilatation of both pupils, sometimes more marked in the one eye than in the other. At this moment the right is larger than the left; but the latter also is abnormally wide. The muscle of accommodation is not much affected on the left side; but on the right side the range of accommodation is reduced one-half: for Jaeger No. 3 can be read only at 16″ with the right eye; while with the left it can be read at 6″. No errors in refraction and no deficiencies in structure were discovered. The muscles of the eye also were intact and their functions normal. There was here some disturbance within the domain of the nervus oculomotorius, which extended, however, only to those nervous branches which preside over the functions of the iris and the ciliary muscle.

It is very probable that the present affection was caused by constitutional syphilis. He had an ulcus syphiliticum induratum, afterward angina and roseola. Several courses of mercurial inunction had caused these and other symptoms to disappear.

The indications were to use iodide of potassium against the supposed primary disease in the nervous tracts and central organs; and to relieve

the local paralysis of the iris and ciliary muscle with ext. fabæ calabar.

The patient did not present himself again for examination.

CASE IX.—*Herpes Zoster along the 1st branch of the Nervus Trigeminus, then Iritis Serosa, afterwards Paralysis of Iris and the Apparatus of Accommodation.*

A merchant, aged 50 years, had during the last six days noticed a hyperæsthesia of the scalp on the left side. Along the tract of the left nerv. trigeminus small vesicles about 1‴ in height and 3‴ in width, containing clear serum, were found upon the upper eyelid, the forehead, and the hairy scalp. The affection soon passed over under an expectative plan of treatment.

After three weeks the patient returned with an iritis serosa. The humor aqueus was cloudy; the membrana Descemetii punctuated with numerous precipitates. This disease was also cured under appropriate therapeutic means within a few months.

Two months after the dismissal of the patient he returned with a new disturbance of vision. A sudden paralysis of the iris and ciliary muscle had occurred. The pupils were dilated; small print was no longer read; Jaeger's No. 5 could only be seen at 20″ distance. The paralysis increased, and Jaeger No. 5 could not be seen at all. After the use of calabar bean Jaeger No. 3 could at last be read. Whether there was a common cause for all these different affections of the visual organs, which followed each other in quick succession, cannot so easily be determined.

CASE X.—*Paralysis of the Ciliary and External Rectus Muscles during Constitutional Syphilis.*

A merchant, 34 years old, is admitted to treatment for a paralysis of the musc. rect. ext. of the right eye, probably the consequence of a former syphilitic infection. Beside the external rectus the ciliary muscle is somewhat affected. The eye is of emmetropic structure, but the range of accommodation is reduced in the right eye as compared with

the left. While with the left eye Jaeger No. 3 is read at 6″—a range of accommodation=$\frac{1}{6}$—the right eye reads No. 3 only at 9″, and has a range of accommodation=$\frac{1}{9}$. The amount of diminution of accommodation of the right eye from that of the left, is therefore $\frac{1}{6}-\frac{1}{9}=\frac{3}{18}-\frac{2}{18}=\frac{1}{18}$; hence, fully one-third of the existing range of accommodation in the healthy eye, which is=$\frac{1}{6}$. In this case, besides the ner. abducens, that part of the ner. oculomotorius which enervates the organs of accommodation, has also been subject to disease, probably in consequence of the general syphilitic infection. The treatment consists in that of constitutional syphilis: a pair of spectacles—on the right side convex No. 18, and on the left plain glass—equalizes the difference of the two eyes, and causes small objects to be perceived by the two eyes at an equal distance of 9″, and thus secures the simultaneous and harmonic action of the dioptric apparatus.

KERATO-CONUS AND ITS TREATMENT.

By Dr. STEINHEIM, OF BIELEFELD.

Translated by Dr. Richard H. Derby, Ophthalmic Surgeon to the Demilt and New York Dispensaries.

(PLATE VII.)

Until latterly, the treatment of the conical malformation of the cornea and its consequences, various as it has been, assumed no fixed and sure form, and the affection, as far as the so greatly reduced vision was concerned, appeared to the patient as well as the surgeon to be incurable. Now, however—thanks to the successful efforts of Prof. Von Graefe—we have found for this form of disease a reliable remedy, securing, even in the most desperate cases, a sufficiently satisfactory improvement, and there can be no doubt that the results will be still better than those we have yet attained, when the operative procedure has been more fully developed.

Various remedies were formerly employed in the treatment of this affection—the pathological anatomy of which was so obscure, and the immediate consequences of which, even where the inner membranes of the eye and the refractive media were otherwise normal—led to so considerable a reduction of vision, that, if absolute

blindness never followed, still the most considerable diminution of the acuity of vision must result. These remedies consisted principally of a small iridectomy, in order to use the apparently normally curved peripherical portions of the cornea for the admission of light; or of iridodesis; or, on the principle of the stenopæic slit, of a diametrical iridodesis. Then, as the development of the conus of the cornea came to be regarded as a consequence of an abnormally increased intra ocular pressure, an iridectomy was recommended, more on the ground that the intraocular pressure would by this step be reduced, than by making a pupil back of such a portion of the cornea as appeared of a more uniform and normally refracting power. On the same principle, and for the same reason, repeated paracentesis of the cornea was made, in order at the same time to improve the conditions of nutrition in the cornea, which evidently in this disease are abnormal. How far all these measures were of any considerable service in the improvement of the vision, on the one hand, or in the reduction of the conus on the other, cannot, from the literature and the reported cases, with certainty be ascertained. This much is clear, that all the measures which were adopted did not satisfy the demands which the surgeon, as well as the patient, had a right to make upon such an operative procedure.

Unsatisfactory as the views upon the cause and nature of kerato-conus were, just as unsatisfactory were the results of the operations that were based upon these

views. For the iridectomy that was intended to reduce the supposed increased intra-ocular pressure, as well as the repeated paracentesis, proved in practice utterly insufficient, and without influence upon the malformation, for, as subsequent studies upon the conditions of tension in the eye showed, the causes of the conical development depended, not upon absolute but upon relatively increased tension, with abnormal thinning of the apex of the cornea. Just as little—although the theory was perfectly correct—did the iridectomy, the iridodesis, and the diametrical iridodesis, improve the acuity of vision, and relieve the patient of those very annoying circles of dispersion and secondary images and colors, carefully as the indications for these operations were laid down by Donders.* Donders says here, in reference to the improvement of the acuity of vision in kerato-conus: "The principal aim of the operation is to make a pupil opposite that portion of the cornea whose curvature is most regular and most closely approaches a spherical or ellipsoidal surface, in order that a distinct image may be formed in the visual line, and especially that direct vision be improved. *A priori*, it is evident that by means of a small pupil the object can be better gained, because the circles of dispersion are then smaller, especially, however, because the smaller the district of the cornea is which is concerned in the production of the image, so much less will the difference be in the refracted rays." Still, notwithstanding all this, in the most favorable

* Archiv. f. Ophth., VII. 1.

cases, where portions of the cornea possessed regular refractive power, the most central and the immediately adjacent portions would exert their disturbing influence of dispersion. Not to speak of the loss of every regularity of refraction of the most refracting portion of the cornea, and the development of a very high degree of irregular astigmatism, there is no portion that refracts the rays in a manner necessary for the formation of the image on the retina, for, not only are the most essential differences in the focal distances of immediately adjacent portions of the cornea to be considered, but the focal points themselves, even of the smallest portions of the refracting surface, are incomplete, and situated in different axes. It is evident enough that these operations, too, which aimed at a better optical condition of the eye with keratoconus, could not attain the practical results which they seemed to promise; moreover, that the iridodesis is an operation which endangers the safety of the eye, through the production of secretory irritation and consequent increased tension, and therefore must be abandoned.

There remained now, for those who with untiring zeal kept fresh in mind the unfortunate condition of these patients, but one way to improve the functions in keratoconus, and that lay in attacking the cornea itself for the improvement of the anomaly of curvature. This great task was accomplished through the repeated efforts of Von Graefe, who first published his method of operating in the Archiv. f. Ophth.,* and later, in the Berliner Klin.

* Archiv. f. Ophth., 12, 2, p. 215.

Wochenschrift,* communicated his additional observations upon the pathology, symptomatology, and treatment of kerato-conus, with several cases. Although the method of operating has been known since that time, still, just as every new measure is reluctantly adopted, often meets with little favor, and sometimes, indeed, is in danger of being regarded as an extravagancy, so this one, up to to-day, has not met with that reception which it really deserves. Ed. Meyer and Horner are, until now, the only ones who have adopted the operation. The last published two cases in Monatsbl. für Augenheilk., 1869, and strongly recommended the operation, while Wecker† writes:

"On ne pourra pas juger définitivement cette méthode, que quand il sera prouvé qu'elle peut arrêter la marche, si souvent progressive, du Kératocône; car sans cela, le malade risquerait de ne gagner par ce traitement qu'un leucome disgracieux."

As an addition to the already published cases, the three following are offered, which, even if they do not essentially differ from the others, still, in their details, and especially in their results, are sufficiently worthy of mention to direct attention again to this method of treatment, and assist in giving it its proper place in the manuals of ophthalmology.

Before I became acquainted with Von Graefe's method of operation and treatment, I had occasion to perform, three times, iridectomy for kerato-conus, but without attaining any considerable improvement.

* Berliner Klin. Wochenschrift, 1868, Nos. 23 and 24.

† Traité des Maladies des Yeux, 1867.

Case I.—In April, 1868, August St., 21 years of age, presented himself; had always enjoyed good health, and had good vision in both eyes. During the last six months he had observed a marked change and deterioration of the vision of the left eye, while the right, until six weeks before, had been perfectly normal. This deterioration had begun and had advanced to its present condition without inflammatory symptoms, without previous disease, and without any assignable cause. The cornea of the left eye presented the picture of an exquisitely developed kerato-conus; on thex ape of the conus there was the well-known gray striate opacity that is always to be seen in highly advanced and completely developed cases. The right eye, as was said above, six weeks before became worse, and now led him to come for advice; on this eye the development of the conus had distinctly commenced, as the profile view and experiments of reflection plainly showed; there was, however, no opacity on the apex, but a very considerable diminution of the acuity of vision. With the left eye he read without a glass Jaeger Nos. 3 and 4 only at a distance of $\frac{3}{4}$ of an inch to an inch, holding the print outwards on account of the nose; a larger type could not be read at a greater distance. With the stenopæic apparatus the acuity of vision was improved so much that he could count fingers at the farthest at a distance of six feet. Glasses were of no service. When he closed the right eye he found the bearings of the room with difficulty, the eye moved in different directions in order to receive the impressions of the separate objects in order. The operation was made on the 20th of May, according to Von Graefe's method. When the artificial corneal ulcer had advanced funnel-shaped so far that only a thin lamella of the cornea was left, a paracentesis was made, and the same repeated during the next six days as often as it became closed, so that during this time a corneal fistula was maintained. Then, when it was evident from the profile view that the conus had become much flatter, differing essentially in curvature from that of the right eye, the ulcer $\frac{3}{4}'''$ in extent was allowed to heal. Notwithstanding a very considerable infiltration about the ulcer the acuity of vision was found on the 19th of June to be very considerably improved, and on the 20th of July the condition was such that with the naked eye the

patient could count fingers when held at a distance of 16′. Finally, six months after the operation, at a time when the result could be regarded as complete, he could read Jaeger No. 1 in 5″, No. 5 in 9″, with −7 Sn. No. 4 in 20′, =S. $\frac{2}{3}\frac{0}{0}$. With the stenopœic slit held horizontally, his vision was still more improved. In contrast to the right eye, whose conus had increased without having reached the same degree as that of the left, the operated eye is now used for all work, and the other, especially in reading, is excluded.

This remarkable result induced me soon after to propose this new method to a patient with kerato-conus, on whom, in 1864, I had made an iridectomy, without, however, any considerable improvement.

Case II.—A. S., mechanic, 37 years old, slightly built, had suffered in his childhood from repeated inflammation of the eyes, as was evident from several light peripherical corneal opacities. At the age of 25 his vision, from no assignable cause, became suddenly so much reduced that he had to give up his occupation as a weaver. In 1864, on account of an advanced kerato-conus of the left eye an iridectomy was made; still, notwithstanding the fact that the new pupil had been made at a favorable point, the vision remained unchanged, and neither glasses nor the stenopæic apparatus gave any improvement. The patient read Jaeger No. 6 at $1\frac{1}{2}$″, and counted fingers with difficulty when held at a distance of 4′. The operation was performed precisely as it is described, but still, through miscalculation of its thickness, the cornea was perforated at a single point. On account of the previous iridectomy a piece of the cornea, $\frac{5}{4}$‴ in extent, and larger than Von Graefe usually advises, was excised. The cauterization with lapis, after the third day, produced the required inflammation; subsequently a paracentesis was made and for several days a fistula maintained, and after the expiration of $2\frac{1}{2}$ weeks the ulcer was allowed to heal. The result was a most favorable one. In the course of the eighth week there appeared on the apex of the conus a circumscribed whitish-gray opacity, a line in diameter; the artificial pupil was perfectly clear, the diminution in the convexity of the cornea so considerable in comparison with that of the other eye, that the patient's friends immediately observed it, and his vision was

so far improved that he read Jaeger No. 1 at 2″ and with $-4\frac{1}{2}$ S was brought up to $\frac{1}{10}$. The patient found his way about easily, and was so well satisfied with the result that he was ready to have the operation performed on the other eye.

Case III.—J. Qu., painter, has always enjoyed good health, but has suffered repeatedly from inflammation of the eyes, between the age of 7 and 14. At the time that he began to draw and paint, in his 17th year, he suddenly became quite myopic, so that he always wore a glass. The left eye was always the weaker of the two, probably on account of considerable corneal opacities, the consequence of inflammation. Up to March, 1849, he had been in the habit of overtaxing his eyes while at his work; then he observed, for the first time, a change in the acuity of his vision; he became polyopic; saw circles of dispersion; the distance between his near and far points grew less, and his glasses were no longer of service. His vision deteriorated so rapidly that he could no longer paint large pictures. In 1853 a tenotomy was performed on the left eye on account of strabismus convergens. Since that time he has observed no considerable change. The kerato-conus has remained the same, while his vision, through constant straining, has grown worse.

Present condition. Patient, who is of a highly nervous organization, has on both eyes an advanced kerato-conus. Ophthalmometrical measurements could not be made, but repeated and various experiments showed in a surprising way the varying size and changing form of the reflections upon different portions of the cornea. Viewed in profile there is a very abrupt and pointed conus, on which can be plainly seen, what has been observed in other cases, that the side of the conical projection, opposite the point of incidence of the rays of light, appears dark as if in shade. On the cornea of both eyes, in addition to this striate grayish opacity on the apex of the conus, are, in its neighborhood, several diffuse spots, the products of previous inflammation. The anterior chamber is deep, the pupil appears variously distorted according as it is regarded from different sides. The ophthalmoscope reveals an extensive sclerectasia post., extending about the optic disk. The apex of the conus offers an abnormally slight resistance to the point of

a sound, is much thinned, and feels like a bladder-like protuberance. The acuity of vision is much less in the left eye than in the right, in consequence of the previously existing strabismus. With the right eye, by holding the print on one side, No. 3 can be read at a distance $\frac{3}{4}''$, but nothing of Nos. 1 and 2; indeed, No. 3 only with difficulty. The largest type cannot be recognized at a greater distance than 3″. By nearly closing the lids No. 1 can be slowly read at $1\frac{1}{2}''$; he can count fingers when they are held at a distance of from 2 to 3 feet; beyond this limit they appear multiplied and surrounded by secondary colors. By placing a finger of his right hand lightly on the upper lid, and exerting in that way a slight pressure on the bulb, at a particular point that he has already found out by practice, the vision is very considerably improved, rising to $\frac{1}{3}$, everything appears distinct, the largest as well as the smallest type appears no longer in circles of dispersion or secondary colors, and, as the patient says, in this way the amount of vision is perfectly satisfactory. Upon examining carefully this experiment, one sees that at the moment the pressure is made the curvature of the cornea changes, that the conus is corrected, and that between the partially closed lids the cornea presents a nearly normal curvature. The patient is in the habit of helping himself in this way while painting. The left eye is much worse than this, and with it he reads only Jæger No. 4 in $\frac{3}{4}''$, and fingers he recognizes when held at a distance of 2′. The vision is not improved with spherical or cylindrical glasses, nor with the stenopæic apparatus. The pressure that raised the vision of the right eye is here of no avail. Patient said that on account of the continually increasing multiplication of the secondary or dispersive images, objects lose their proper form, and assume monstrous and irregular shapes.

On the 24th October, 1869, Von Graefe's operation was performed on the left eye, a portion of the apex of the conus, $\frac{3}{4}'''$ in extent, was excised with great care; during the next few days the surface of the corneal wound was repeatedly cauterized with lapis; when the cornea at this point was reduced to a very thin lamella, paracentesis was made, and for several days a corneal fistula was maintained. With-

out entering into details and mentioning one by one the little modifications which may be indicated in the course of the case, it is enough to say here—that I attach especial importance to the establishing a fistula for some time; further, that I am in the habit of encouraging a thinning of the base of the ulcer, not funnel-shaped, but over a considerable portion of its extent; in this way the paracentesis can be repeated at intervals at different points of the base of the ulcer, and thus a firmer cicatricial contraction not only of the outer lamellæ of the cornea, but what is also of importance, a correspondingly firm contraction of the deeper lamellæ be secured. In this case, notwithstanding the great irritability of the patient, the process of healing advanced very favorably; at the end of the 4th week, in spite of the infiltration of the cornea about the ulcer, the acuity of vision had considerably improved, and at the end of the 8th week the examination showed a very marked improvement: he could see plainly, without circles of dispersion, the hand held at a distance of 7′–8′, with $-4\frac{1}{2}$, fingers in 18′, Jaeger No. 1 in 4″, No. 4 in 5″, distinctly; with $-4\frac{1}{2}$′, No. 14 in 11″; $S=\frac{16}{200}$. The addition of a —cylind. glass brought S up a little.

In order to watch the progress of his condition more accurately, the patient drew *a series of sketches* showing how a point, a line, a circle, a star-shaped, or sickle-shaped figure, and the crescent in the sky appeared to him prior to the operation. He represented these very objects again in the second month of his cure. The diagrams show a striking difference, and illustrate clearly the improvement in the visual functions. For this reason I may be allowed to reproduce these sketches.

EXPLANATION OF THE DRAWINGS OF TABLE VII.

Figs. 1, 2, 3, 4, 5 appeared before the operation at a distance of 25 centimetres as in *Figs.* 6, 7, 8, 9, 10;

Eight weeks subsequently as in *Figs.* 11, 12, 13, 14, 15.

After a complete cure had been effected a considerable portion of the dispersion circles disappeared.

Fig. 14. Circle at a distance of 25 ctms.

Fig. 16. Circle at a distance of 50 ctms.

Fig. 17. Crescent in the sky before the operation.
Fig. 18. Crescent in the fifth week after the operation.
Fig. 19. Crescent,—ninth week after the operation.
Fig. 20. Crescent—lids partially closed.

ON THE EMPLOYMENT OF THE NASAL DOUCHE.

BY L. ELSBERG, M.D.,

Clinical Prof. of Diseases of the Throat in the University of New York.

A NUMBER of cases of otitis media have recently been published, said to have been caused by the use of the nasal douche. Aurists have come to regard the method as fraught with "serious danger," some going so far as to interdict it altogether. I believe Prof. Roosa is right in stating that his case was the first published "where the use of the nasal douche was considered the cause of the aural inflammation." Prof. Moos has reported injurious consequences following it. Prof. Knapp and Dr. Pardee join in the condemnation. Personally convinced that, when properly used, the douche is both harmless and useful in the treatment of diseases of the pharyngo-nasal space, I deem it not unimportant to say a few words in its favor. The danger dreaded is from the passage of liquid into the tympanum, and this alone, perhaps, comes properly within the scope of the ARCHIVES. I shall therefore address myself to this point only.

In the first place, the number of adult persons in whom, when the parts are at rest (*i. e.*, when the persons are not engaged in the act of swallowing, or the

like), the pharyngeal end of the Eustachian tube is naturally patent, and not sufficiently far back to make the accident possible, is very small. Rhinoscopical examinations, to say nothing of anatomical investigation, demonstrate this so indubitably, that it can be asserted dogmatically. In disease, the mucous membrane lining the Eustachian tubes, and surrounding their pharyngeal orifices, so frequently becomes swollen, that the cases in which the accident under the same circumstances otherwise would be possible, become still fewer. Indeed, I have several times, since Prof. Roosa's article has been published in these ARCHIVES, made the careful trial, and found it difficult in cases of chronic catarrh to make fluids enter by means of the douche, even during swallowing, although the Eustachian tubes appeared free, and there was not the slightest difficulty of hearing present. That I do not mean to allude, in this connection, to cases in which the Eustachian tubes are abnormally impermeable, is, of course, understood; nor do I here lay any stress upon the fact that I have more than once, without the least injurious consequences, injected liquid into the Eustachian tube, both through the Eustachian catheter, by a syringe, and through the Eustachian nebulizer, as well as by means of the douche, during deglutition. But I believe that, in the rare cases in which the occurrence of the accident would, so far as the patency of the tube is concerned, be possible without deglutition, it is made very improbable when the douche is properly employed.

In the next place, let me refer to my experience. Of

1,600 different individuals who have, during the past six years, used the douche under my direct advice, the following four have experienced unpleasant ear-symptoms:

I. Dr. Med. J. L., æt. 58 years, a practising dentist of Paterson, New Jersey, had long been suffering with chronic catarrh of the pharynx and nose; had cough, obstructed breathing (especially through left nostril), hypersecretion of phlegm, senses of smell and taste dulled, hearing unaffected. On examination, thickened membrane and some erosions were found. In addition to appropriate general and local treatment, he was ordered to use the nasal douche with tepid salt water, for cleansing purposes, twice daily. After the lapse of 3 or 4 weeks, he had improved, as he expressed it, "wonderfully," when—I am quoting his words from my note-book—he "attempted on my (his) own hook to combine with closed mouth Valsalva's experiment with the douche." He "suddenly felt the salt water enter the ears with a great deal of force, like the rushing of Niagara, and almost fainted." I saw him 3 days later, when he complained of fulness, pain, and throbbing in the ears, and was *entirely deaf*. These symptoms had set in immediately after his "experiment," and the pain in the head had been so intense that he was forced to remain in bed. He bore neither a rhinoscopical examination, nor even a thorough inspection of the external meatus, and I limited myself to giving him a pantomimic scolding, and an anodyne hypodermic injection. The symptoms subsided gradually; he did not leave the house, however, for 10 days longer. When he came to my office again, he was free from pain and the other unpleasant sensations about the head caused by the experiment, and his hearing was normal again. We recommenced the douche, which he used regularly twice daily for 4 months longer, until his final cure.

II. D. R. A., æt. 30 years, a member of the Chicago gambling fraternity, had had purulent otitis a number of times, from his account, and ozœnic catarrh of the head for many years. He denied ever having had syphilis. Nothing had given him so much relief as the douche, which he had been using for nearly a year, and which I ordered him to

continue. I found in the pharyngo-nasal space several patches of severe ulceration, although the bone seemed unaffected. Cleansing and cicatrization appeared to set in under local applications, when, after only a few weeks' treatment, after an exposure, he was one night taken with what he called one of his old ear-attacks. This he ascribed at first to a very forcible blowing of his nose, but afterwards to too severe a use of the douche. My own belief is that he was out on a spree that night, and did not know what he had done. This was, however, the first attack of otitis he had had during the year of using the douche, while he had had them previously. I passed him from my care to that of Prof. Holcomb, who attended him during a most severe purulent otitis, requiring abscess-opening in the mastoid region, etc.

III. Mr. E., æt. 32 years, came to me with large syphilitic ulcers in the mouth, involving especially the tonsil and both the anterior and posterior arches of the palate on the left side, which ulcers, under general antisyphilitic treatment and local applications, rapidly cicatrized so as to be perfectly healed in about six weeks. But he had had scarlet fever three times in his life, and from childhood his ears had troubled him from time to time. In addition, catarrh of the pharyngo-nasal space is quite prevalent in his family, I having treated his sister for this affection, and he himself and other members suffering from it. I recommended him to use the douche as a palliative, and soon after I had dismissed him from treatment of his mouth, he sent for me one evening on account of a pain in his ear caused by the use of the douche with cold water, and the neglect of his usual precautions. This was in winter, and the water quite cold. I placed him under the care of Prof. Knapp, and this is the case reported in these ARCHIVES, No. 2, p. 366, in connection with which Prof. Knapp made those beautiful observations on the perception of double tones, or "diplacusis binauricularis," as he proposes the disease should be called.

IV. Mrs. B. had long been suffering from follicular disease of the whole pharyngo-pulmonary mucous membrane, and also from slight nasal catarrh, and increasing difficulty of hearing. From time to time she had purulent discharge from the ears, and her membranæ tympanorum

were found perforated. During a long course of treatment I tried several times to get her used to the nasal douche, but after using it a little while it would cause so much irritation in the neighborhood of the Eustachian tubes that it had to be discontinued. Whenever she could get the liquid to come out of the outer ears she could bear it best.

Other patients have occasionally complained of unpleasant sensations in the ears, particularly on using the douche awkwardly, but in only the four cases mentioned have such symptoms amounted to anything severe, or lasted longer than from half an hour to a few hours. It is remarkable that I have never seen any severe ear symptoms follow the douche in children, although I have employed it from the age of six years upward, and in one case at the age of four years. With that one exception, I have always used, in children younger than six years, the Davidson or another syringe through the nose from the front. In addition to these sixteen hundred, a large number of persons whom I have never seen as patients have learned the regular use of the douche from these. Nasal and post-nasal catarrh is very common in this country, and unless particularly troublesome, it is not brought directly to the notice of the physician, although the sufferers are anxious enough to do something for palliation, and many members of families so situated that they cannot obtain regular treatment, or who are constantly exposed to the causes of catarrhal affections, use the douche by themselves; and, strange as it may appear, I know of households, the unaffected members of which have adopted the occasional use of the

douche as a matter of cleanliness and toilet. Yet from this additional large though uncertain number I have never heard of any case in which bad symptoms followed the use of the douche.

To my certain knowledge, some have continued it once or twice daily for a number of years, neglecting it, perhaps, not more than half a dozen times a year; and many have for months and months. This is my answer to Prof. Roosa's statement (ARCHIVES, p. 363): "I have seen few cases where the use of the douche could be tolerated for any length of time. Its daily employment is very often attended with great discomfort."

Dr. J. Solis Cohen, of Philadelphia, has informed me that his experience corresponds with mine.

I have insisted upon the harmlessness of the nasal douche *when properly employed.* Of course it may do harm when improperly used. But it need not be supposed that the necessary precautions are complicated or difficult to take. On the contrary, they are so simple that they can be safely left to patients after they are made to understand them. Without discussing the method fully, or the various medicinal solutions, I will speak only of three or four points which apply to the douche when used for general rinsing, viz.: 1. Ordinary water, even distilled water, does not act so soothingly or indifferently as water containing a teaspoonful, or a little more, of common salt to the pint. 2. The temperature must be nearly that of blood-heat, though warmer solutions are frequently, though not always, well borne; *cold*

liquids hardly ever are. 3. Involuntary swallowing during the operation must be prevented, therefore, the mouth must always be opened, and the tongue should be held outside of the open mouth by the hand, covered with a towel, glove, or the like; if liquid comes into the throat, which is to be swallowed or ejected, the operation must be interrupted; and, 4. The pressure must be but slight at first, *i. e.*, the vessel containing the liquid must not stand too high above the level of the nostril, but may be gradually raised.

If it be deemed necessary to analyze the cases of accident published, it may be seen that Roosa's case is uncertain. Knapp's case is one of my four: the medical student "neglected to open his mouth as widely as usual, a drop trickled down the throat, and involuntary swallowing took place;" and Pardee's other case rests only upon "the intelligent and straightforward statement of the patient," and also leaves it uncertain whether the douche was properly used or not. My cases, besides being so small a percentage, explain themselves.

That the douche is but one means, and not to take the place of other means, in the hands of the physician, I presume I need hardly add. The only point I wanted to make is, that the douche, so far as it goes, is both efficient and safe.

TWO CASES OF EMPHYSEMA OF THE ORBIT, EYELIDS, AND ADJACENT PARTS.

—

REPORTED BY DR. E. GRUENING, OF NEW YORK,

Assistant Surgeon to the New York Ophthalmic and Aural Institute.

CASE I.—*Emphysema of the Orbit and Eyelids.—Exophthalmus.*

August Stark, 30 years of age, received a severe blow on the left side of his forehead, on the evening of August 29th, 1870. He did not feel much pain at the time, and slept well all night. On waking next morning he found the left upper eyelid somewhat swollen and bloodshot. While washing his face he drew up some water through the nostrils and then *blew his nose*, whereupon the right *eyeball protruded*, and both eyelids swelled considerably. He suffered no pain. The patient came to the clinic that very day—eighteen hours after he had received the injury. On examination, the right upper and lower eyelids were very much distended; the palpebral fissure was closed. By voluntary efforts the patient was unable to open the eye. The swelling was soft and elastic, not tender to the touch, and yielded an emphysematous *crackling* on palpation. The left superciliary region was also swollen, but here the tumor was tender and resistant to the touch, the tenderness being intense over the supra-orbital notch. The left upper lid was perfectly movable. By snuffing, the patient drew coagulated blood from the posterior nasal region. On separating forcibly the lids of the right eye the globe was seen to protrude, its cornea occupying a lower plane than that of the left eye. Crossed diplopia appeared in the whole extent of the visual field. Beyond the horizontal plane the images showed both

a lateral and vertical separation. With the ophthalmoscope no lesion of the interior of the eye could be detected. Vision was perfect with either eye.

The patient was advised to apply compresses, dipped in tincture of arnica, over the swelling; to refrain from blowing his nose, and to come back the following day. He did not return, most probably on account of the rapid diminution of the swelling.

CASE II.—*Emphysema of the Eyelids and Adjacent Parts.*

John Schneyder, aged 35, on being admitted into the New York Ophthalmic and Aural Institute, January 5, 1871, gave the following history: Four days before, he had jumped from a railroad carriage in motion, fallen, struck the right side of his forehead, and become unconscious. He regained his senses after fifteen minutes, and then found that both lids of the right eye were enlarged to such an extent as to cover the globe completely. His face and clothes were stained with blood, which could be traced from the nostrils. He then *blew his nose*, and experienced a *peculiar creeping* and painful sensation over *the right temple* and behind the corresponding ear, and at the same time noticed that his *eyelids* had become still *more enlarged*, and the neighboring parts considerably distended.

On examination, four days after the accident, the lids of the right eye were tense and shining, and projected beyond the superciliary ridge. They were immovable by a voluntary effort, but permitted a forcible separation of about 1‴. The temporal and post-aural regions also exhibited a swelling, the measurements of which were taken as follows:—Antero-posterior diameter extending from the outer commissure to a point behind the auricle=4″; Vertical diameter extending upward from the middle of the right zygomatic arch=2″; Summit, situated midway between the outer border of the orbit and the auricle, projecting beyond the surface of the temple = ¾″. The skin of the parts was not discolored, and its temperature not perceptibly increased. On palpation an emphysematous crepitation was distinctly felt everywhere.

The patient complained of no pain, except when pressure was made at the inner canthus.

A compressive bandage was applied over the swelling, and the patient told to abstain from blowing his nose. After four days the swelling had disappeared, and the patient was discharged cured.

Cases of emphysema of the eyelids and their neighboring parts, occasioned by a fracture of the inner wall of the orbit, are sufficiently rare to warrant their publication. The *diagnosis* of this condition is readily made, when the unmistakable *crepitation* is felt. The injury received, and the *sudden appearance of the swelling after an expiratory effort* with mouth and nostrils closed, also serve to confirm the diagnosis. In the cases under consideration the fracture of the inner wall of the orbit must have been produced by contrecoup. The injury was inflicted at a distance from the seat of the fracture, and the soft parts covering the latter showed no signs of direct violence. From the existence of exophthalmus in the first case we may infer that the fracture implicated the posterior portion of the inner wall of the orbit—the os planum of the ethmoid. As to the second case, the seat of fracture would most probably have been found more anteriorly, perhaps in the lachrymal bone. A remarkable feature of this case was the considerable extent of the emphysema.

The treatment of such cases is very simple. A compressive bandage should be applied over the swelling, and the patient directed to refrain from blowing his nose. The observance of this restriction will generally prove sufficient to effect a cure within a few days.

CONTRIBUTIONS TO THE CLINICAL HISTORY OF INJURIES OF THE EYEBALL AND ITS SURROUNDINGS.

—

By Dr. JOSEPH AUB,

Assistant Surgeon to the New York Ophthalmic and Aural Institute.

I.

Unsuspected presence of a Foreign Body near the Apex of the Orbit—Successful Removal three weeks after the injury.

Mr. P., aged 24, was struck, October 4th, 1869, in the left eye by a spike discharged from an air-gun held at a distance of 3′ from his face. The eye immediately discharged much fluid and was very painful. The lids were greatly swollen and sight was reduced to perception of light. Mr. P. and the bystanders asserted that the projectile, after striking the eye, was thrown off and lost. Cold applications were made for two days to reduce the swelling, but without success. I was called to see him two days after the injury. I found the lids very much swollen; the conjunctiva was very much injected and chemosed; a portion of the conjunctiva was protruding between the partially closed lids. On separating the lids with Desmarres' elevateurs I found a large perforating wound of the cornea. Some remnants of the cornea, which were purulently infiltrated, were still visible, and were in part covered by the iris which was turned back over them. No trace of the lens was to be seen. The vitreous had almost entirely escaped, and the vitreous space was filled with a dirty yellowish mass, mixed with what seemed to be short hairs. Future examination, however, proved that these were woollen fibres. Warm applications were ordered to accelerate the suppurative process, since all hope of saving the eye was gone. Morphine was given

internally and also hypodermically, to still the pain. I saw the patient every other day for two weeks; suppuration was progressing favorably, and at the end of this time the lids had become reduced, the chemosis of the conjunctiva was almost gone, and the eyeball was shrunk to about one-half its original size. The pus which had been discharged during these two weeks, however, had continually an admixture of these woollen fibres, so that I concluded, notwithstanding the assertion of the patient to the contrary, that the projectile was still in the eye or orbit. The most careful examination of the eye with a probe led to no corroboration of my opinion. Seventeen days after the injury the patient came to the New York Ophthalmic and Aural Institute, and another careful examination was made. The eyeball was now reduced to about one-third its normal size, and was pressed towards the upper and inner portion of the orbit. A small opening in the conjunctiva gave free exit to the pus from the orbit, which was still mixed with the woollen threads. After carefully probing, Dr. Knapp found a hard substance at the outer and lower portion of the orbit near its apex. It proved to be encapsuled and firmly adherent to these parts. Three days after a second examination was made, and the presence of the hard body being corroborated, its removal was decided upon.

The patient was anæsthetized, and, after making the conjunctival opening larger, the foreign body was loosened from its adhesions and extracted with a pair of forceps. The wound continued suppurating for four days after the removal, and then closed permanently. The patient has now a very good stump, and bears an artificial eye with ease. The projectile measured, from the point, which was cast-steel, to the end of the still remaining woollen fibres, 21 mm., and in its widest part 6 mm. Its original length, with the woollen fibres intact, was 32 mm.

The projectile must have pierced the cornea, all the refracting media and the sclerotic, and lodged with its point in the orbit, the posterior scleral wound being filled up by the end of the woollen fibres. As suppuration progressed and the eyeball shrank, the anterior and posterior scleral wounds became closed and the pus formed an exit through the conjunctiva, pushing the eyeball aside.

This case seems of practical importance, showing the necessity of a careful examination of the entire orbit, even if the patient asserts that the foreign body came out and the symptoms of irritation diminish in such a manner as to render his assertion probable. We may urge this precaution so much the more, as cases are on record in which foreign bodies lodged unsuspected behind the eyeball in the orbit, and caused death by producing purulent basilar meningitis, continued from the original focus of the suppuration at the apex of the orbit.

II.

Two Cases of Traumatic Suppurative Choroiditis, one ending in Recovery, the other in Destruction of Sight.

G. S., aged 20, came to the N. Y. Ophthalmic and Aural Institute, on the 10th of September, 1870. He was struck on the outer side of the right eye, two days previous, by a piece of steel. A portion of the steel was removed at the time, but there was still a piece remaining. He had great pain in the eye during the night, no sleep and some fever. The eye showed some swelling of the conjunctiva, especially over the tendon of the external rectus muscle, where a small piece of steel was still situated. This was immediately removed. At the outer side of the cornea, a large ulcer was to be seen with clear edges. Circumcorneal injection was well marked. Iris was swollen, and purulently infiltrated, pupil narrow and hypopion in the anterior chamber. An exudation in the pupillary space prevented an examination of the details of the fundus. Sight was reduced to counting fingers in one foot distance. F. was entire. The patient was admitted to the Institution. Six leeches were applied to the temple. A four-grain solution of atropine was instilled into the eye every hour and an aperient remedy given. The patient was somewhat improved next day, and the pupil partially dilated. The day following, however, he had more fever, the

eye was more injected, and the chemosis increased. Pupil was more contracted, the iris more swollen, and the anterior chamber diffusedly opaque. Hypopion had increased in size. Treatment being still continued, the patient's condition commenced to improve on the 4th day after his admission to the hospital. Pupil commenced dilating; the turbidity of the contents of the anterior chamber was diminished. The chemosis, however, increased, as also the swelling over the tendon of the external rectus. But very limited movements outwards were possible. Through the now partially free pupil, the fundus could be seen to be covered in its outer and lower portions by a yellowish exudation, which could be clearly demonstrated by oblique light. F. was imperfect in portions corresponding to the exudation. The following day the swelling over the external muscle had increased, and at its summit a small black spot made its appearanoe, which proved to be the end of a piece of steel perforating the coats of the eye. It was seized with a pair of forceps and removed. Its length was 2‴ and its width 1‴. From this day the eye improved. The swelling of the conjunctiva gradually subsided. Ulcer of cornea healed rapidly. Movements outwards were again possible. The pupil became ad maximum dilated. The hypopion in the anterior chamber disappeared, and at the same time the exudation in the fundus became gradually absorbed. Vision improved rapidly, so that on the eleventh day after the injury the patient counted fingers in fifteen feet, and the visual field was again entirely restored. S. was then discharged from the hospital. One month after the injury, S=$\frac{20}{20}$, and with some exertion the patient could read Jaeger 1½ in 10″.

A marked contrast to the favorable termination of the preceding case is furnished by the following:—

George McG., aged 34, carpenter by trade, was struck, October 22d, by a nail, on the inner surface of the left eye, about 2‴ behind the corneo-scleral margin, causing a ragged perforating wound of the sclerotic and inner membranes. About one hour after the accident, he presented himself at the clinic and was examined by Dr. Gruening, who advised

him to enter the hospital and submit to treatment. At this examination his vision was pretty good, and F. was perfect, but he complained of great pain and intolerance of light. Since the patient refused to enter the hospital, he was advised by the doctor to have leeches applied to the temple, to make instillations of atropine every two hours, and to remain in bed, and in a darkened room. He was asked to show himself again on the third day following. The eye then presented the following appearance: Lids normal, eyeball slightly injected and somewhat chemosed; cornea and ant. chamber as well as the lens perfectly clear. Iris healthy, and the pupil fully dilated. The external wound was covered with conjunctival tissue. From the inner surface of the perforating wound yellowish bands were seen stretching in all directions through the vitreous body. Vision was reduced to the perception of light. F. was wanting outwards. The prognosis as to sight was very bad. The patient was told that the eye, if allowed to remain, would undoubtedly undergo a panophthalmitic process, and then shrink, no sight remaining, and the choice was left to him to have it removed or not. He preferred the latter. The lids became swollen, eye injected and chemosed, the chemosed portion protruding between the lids; in short, the suppurative process took its usual course until the twentieth day after the injury, when Dr. Knapp made a paracentesis of the vitreous chamber, for the purpose of relieving the intense pain caused by the increased tension. The relief was almost instantaneous, and from this time on the more violent symptoms abated. The eye gradually became smaller, and at the present time, two months after the injury, it is about one-half its usual size. Cornea and anterior chamber as well as the lens are clear. The pupil is large, and the iris becoming atrophic. The eye is entirely free from any irritation whatever. No perception of light.

III.

A Case of Rupture of the Choroid at the Macula Lutea, of the Choroid and Retina at the Ora Serrata, and of the Posterior Capsule of the Lens.

John D. H., aged 14, consulted me June 15th, 1870. On the 7th instant a piece of wood, thrown with considerable force, struck his left

eye, at the outer side, causing an immediate swelling of the eyelids and inflammation of the eyeball, combined with great pain. Cold applications were immediately made, and the day following he was enabled to open the eyelids. The eyeball was still very red, and vision was reduced to the perception of light. Under the constant use of cold applications the external symptoms were reduced, and when brought to me the eye presented the following state:—Upper lid slightly swollen and heavy; eyeball white. Focal illumination shows the cornea and anterior chamber to be clear and the iris normal in color and glistening, but reacting very slowly to light. The anterior capsule and the substance of the lens, as far as can be demonstrated by oblique light, seem also to be clear. Ophthalmoscopic examinations, however, showed an opacity of the lens at its posterior pole, and the vitreous so diffusedly opaque as to render an examination of the fundus oculi impossible. After dilating the pupil with atropine I observed that the opacity at the posterior pole of the lens was due to a rupture of the posterior capsule and a commencing opacification of the lens-fibres. In the vitreous body I could see several floating particles, evidently coagulated blood, and at the anterior portion of the fundus, to the extreme left, a dark mass was distinguished, one end of which floated freely in the vitreous whilst the other end was attached to the fundus in that region. Other details of the background could not be observed. Vision was reduced to counting fingers at a distance of 6″ in the lower and outer portion of the visual field. I ordered six leeches to be applied to the temple and the bleeding to be kept up for two hours; instillation of a four-grain solution of atropine three times daily; and inunctions with mercurial ointment, combined with some extract of opium, in the forehead and temple twice daily. The patient was told to remain quietly in a darkened room, and to show himself again in one week.

June 21st, two weeks after the accident, pain had ceased. Eyeball was white. Pupil was fully dilated. The opacity of the lens was somewhat increased in size, and the lens-fibres seemed to be protruding into the vitreous space and thereby increasing the rupture of the posterior capsule. Ophthalmoscopic examination shows the vitreous body

less opaque, the floating particles, with the exception of the dark mass seen at the former examination attached to the fundus, were reduced in size. This dark mass, which proved to be coagulated blood, had detached itself from the fundus, and, floating about in the vitreous, at times completely obscured vision. The papilla could be plainly seen. The inner half was normal, whilst the outer half was covered by a thin layer of blood extending towards the macula lutea. Between the papilla and the fovea centralis one large patch of blood was situated, whilst below the papilla were several smaller clots lying along the course of the vessels. At the macula lutea itself was another collection of blood which did not seem to have any direct connection with the blood at the papilla. At the anterior and outer portion of the fundus, at the junction of the choroid proper with the ciliary portion of the uveal tract, a gap was seen extending upwards and backwards. The borders of this gaping portion were lined with pigment. The rest of the fundus was normal in appearance. Vision had increased to $\frac{3}{200}$. F. the same. Treatment was continued in the same manner. The fundus gradually became more clearly visible, and as the blood became absorbed, an isolated rupture of the choroid, having two offsets above and three below, could be seen in the region of the macula. The borders were lined with pigment and sharply defined. The blood-clot at the outer side of the papilla had become absorbed, leaving a white atrophic patch in the fundus, between the papilla and the macula. The rupture of the anterior portion of the fundus was now filled with cicatricial tissue, the borders being still lined with pigment. S. was increased to $\frac{6}{200}$. F. the same as before. The opacity of the posterior portion of the lens gradually increased from this time until the 10th of October, thereby impairing sight until it was reduced to the perception of light. Discision was then made without any inflammation following, and a good portion of the lens absorbed. On the 3d of November the lens was again punctured. The opening of the capsule was somewhat larger this time, and some reaction followed on the part of the iris. The patient recovered, however, within five days, with one synechia at the upper and outer portion. The lens had become so much absorbed as to allow of some sight. He

counted fingers at 2′ in the outer and lower portion of visual field.

Absorption of the lens matter progressed favorably, and on the 14th of March, 1871, the eye presented the following appearance: Eyeball white, cornea clear, pupil irregularly dilated, owing to two posterior synechiæ, one in the upper and outer and the other in the lower and outer quadrant. The pupillary space was filled with the opaque capsule which admitted light through a very small central opening. Vitreous was slightly opaque; no details of the fundus could be seen. $S=\frac{5}{200}$.

It was determined to enlarge the central opening of the capsule, which was done in the following manner: A needle for the laceration of capsular opacities was passed through the cornea at the inner end of the horizontal meridian, and pushed forward through the opening in the secondary cataract. A similar needle was then passed through the cornea at the outer end of the horizontal meridian. The last needle was also passed through the small opening in the capsule in such a manner as to cross the first needle almost at a right angle. The needles were then separated, thereby forcibly dilating the opening and leaving a large and clear central pupil. Very little reaction followed this operation, and patient was discharged in four days. Patient can now count fingers in 12′ in the lower and outer portion of visual field.

This case is of special interest, since it is the only one on record in which a simultaneous rupture of the choroid at the macula and at the periphery existed. That the retina and choroid were torn at the ora serrata is proven by the fact that the choroidal hemorrhage escaped freely into the vitreous and left no blood collected between the choroid and retina. The rupture at the macula can only be accounted for by contre-coup, whilst the rupture at the periphery and of the posterior lens-capsule must be accepted as the direct effect of the injury. That the entire upper and inner part of the visual field should be

wanting is explained by the inflammatory changes in the retina, which usually follow choroidal rupture, and in which occasionally the entire thickness of the retina becomes implicated. The rupture being at the macula lutea, all that portion of the retina to the outer side became affected by the inflammatory process, and vision in the corresponding portion of the visual field was, in consequence thereof, lost.

IV.

Traumatic Dislocation of the Crystalline Lens into the Vitreous Chamber, with Preservation of Good Sight.

Agnes L., aged 72, came to the clinic in May, 1870. She says that about two years previous she was struck by a piece of wood on the upper lid of the right eye. The eye was immediately much inflamed and swollen, and to relieve this she made cold applications. Vision was reduced to perception of light, in which state it remained for three months. Then the sight commenced to improve, and has continued so doing up to the present time. She can at present distinguish objects at a pretty good distance, and counts fingers in six feet. The left eye had been used for reading and sewing until, within three months, the sight commenced failing, and has been decreasing ever since. She came to inquire whether anything could be done to improve the sight of the injured eye.

Examination of the left eye showed an immature cataract with good visual field, and S. $= \frac{3}{200}$. The right eye was free from all inflammatory symptoms. The cornea, as well as the anterior chamber, clear; pupil large; iris healthy, but tremulous. The ophthalmoscopic examination showed no opacity of the vitreous body. At the bottom of the fundus, which was normal in all other portions, a round semi-transparent body was seen, which seemed to be attached anteriorly to the ciliary body and inferiorly to the retina. It was retained in its position by these attachments, no matter how much or how rapidly the eye was moved. This proved to be the crystalline lens, which had been dislocated into the

vitreous body by the injury two years previously, during which time, however, it had, at least partially, retained its transparency, allowing that portion of the fundus on which it rested to be illuminated by light passing through it. The tension of the eyeball was normal.

On testing the eye with strong convex glasses we found S.with convex $3\frac{1}{2} = \frac{20}{40}$, and the patient could read Jaeger 1 with convex $2\frac{1}{2}$ fluently in eight inches. These glasses were accordingly ordered, and rendered vision excellent in the eye which the patient had formerly considered useless.

V.

Eye Hurt by a Gunshot—No Perceptible Lesion—Remarkable Disturbance of Sight—Perfect Recovery.

H. W., aged 35, consulted me November 28th. He states that on Thanksgiving Day, whilst on a hunting expedition with some friends, the gun of one of the party standing at some distance from him was accidentally discharged, several of the shot striking his left eye and the surrounding parts. Two shot were removed from the forehead and four from the upper lid immediately after the accident, whilst the patient himself removed one from the outer portion of the conjunctiva. W. was not aware whether any had penetrated the eye or not. The swelling of the lids and the redness of the eyeball, which immediately followed the injury, were promptly reduced by the judicious use of cold applications. When I saw him three days afterward, the eye presented the following appearance: Upper eyelid at its ciliary margin was somewhat swollen and showed the impressions made by the shot, none of which had perforated the lid. The eyeball and conjunctiva of the lids were injected and slightly swollen. Circumcorneal injection very much pronounced. Cornea clear; anterior chamber half filled with bloody serum. Pupil contracted, iris hyperæmic and of a greenish tint. At the upper and outer portion the iris bulged, the projecting portion being equal to about one-sixth of the entire circumferencc. Details of the fundus not discernible. After instilling atropine, the pupil dilated partially. The lens proved to be intact, but its upper and outer portion was pushed from its proper position towards the cornea, and caused the prominence

of the iris above mentioned. The vitreous body was diffusedly opaque. No details of the background visible. Sight was reduced to counting fingers in four feet. Visual field was perfect. Tension of the eye slightly diminished, and pressure on the upper portion near the equator was painful, but on closest examination, I could detect no lesion to account for this pain. Application of leeches to the temple and rest in bed, with the instillation of atropine and the use of mercurial ointment twice daily, were ordered.

When the patient called again, three days later, his eyeball was almost white, cornea clear, anterior chamber perfectly free from blood, iris normal in color, pupil ad maximum dilated, the upper and outer portion of the iris still being pushed forward by the protruding portion of the lens. The vitreous body was much clearer, and all the details of the fundus oculi could be seen. Nothing abnormal was found, except a slightly hyperæmic condition of the choroid. The patient experienced a remarkable kind of metamorphopsia. When he held a straight staff diagonally before his injured eye, the end directed upward and outward appeared thicker, whereas the lower and inner end appeared thinner than its normal size. In turning the staff upon his visual line, the abnormal appearance gradually diminished, and it disappeared entirely when the staff had assumed a position at right angles to its first position. Sight was increased to counting fingers in twelve feet without glasses, and with a concave cylindrical glass of twenty inches focal distance, and the upper end of the axis turned about 20° inwards, S. $= \frac{20}{100}$. The patient was advised to continue the atropine and the inunctions, and to keep himself quiet in a dark room.

Six days after the last examination the patient again presented himself, and the eye was found to be free from all inflammation, the lens and vitreous perfectly clear, the pupil ad maximum dilated, whilst the iris had lost its prominence at the upper and outer portion. The patient could now notice no difference in the appearance of the ends of a straight staff, and on testing him with glasses, I found that the weakest cylindrical glasses reduced vision, whilst with concave 26 S. was equal to $\frac{20}{50}$. Without glasses S. with the injured eye was equal to $\frac{20}{50}$. All

treatment was now discontinued, and when the patient again presented himself on the 18th of December, twenty-four days after the injury, his acuity of vision was $\frac{20}{20}$, his refraction emmetropic, and his accommodation perfect.

This case presents several very peculiar features, in explanation of which I may be allowed to make some remarks. The bulging of the iris can only have been produced either by an incomplete dislocation of the lens or a rupture, or, at least, a relaxation of the corresponding part of its suspensory ligament, allowing the lens, by the unchecked elasticity of its capsule, to assume a more spherical shape in that region. The incomplete dislocation could not, it seems to me, explain the metamorphopsia, which,on the other hand, may well have been caused by the partial increase of curvature and thickness of the lens, due to the partial rupture of the zonula, in the following manner: The optical system had become myopic in the outer and upper portion where the swelling of the lens existed, and the whole system must have had assumed more or less the shape and the action of a conical lens, the axis of which was turned diagonally from above and outwards to below and inwards. The image of a straight linear object held parallel to the axis of this conical lens must have had, as I have convinced myself by experiments, a tapering form on the retina, the upper and outer end of which was smaller than the lower and inner end. The thicker end of the image was also more indistinct, in consequence of the dispersion circles from the myopic portion of the lens.

THE DIAGNOSIS OF COMPLETE PARALYSIS OF THE AUDITORY NERVE. A CONTRIBUTION TO THE PATHOLOGY OF THE MEDULLA OBLONGATA.

By Prof. S. MOOS, M.D.

Translated by E. Gruening, M.D.

On the 12th of December, 1869, Drs. Picot and Deimling consulted me with regard to the following case:

A soldier, who had served actively for three months and enjoyed good health up to three weeks ago, was, at that time, suddenly attacked with syncope, preceded by vertigo. Prior to this attack he had at various times complained of dizziness. The patient fell, became unconscious, and on recovery found himself completely deaf to speech, noises, and tones. At the same time he heard a loud humming noise, which gradually diminished and finally disappeared. On examination the patient presented a severe nasal and slight tubal catarrh, diminished sensibility of the facial skin and the ocular conjunctiva, and anæsthesia of the drumheads.

A very intense galvanic current (30 Stöhrer's elements—internal arrangement), turned in rapid succession from 15 to 20 times, produced neither auditory nor other sensations, though it caused twitching of the muscles of the right upper extremity. Sensation of light could be obtained with powerful currents only; sensation of taste with comparatively weak currents. The muscles of the extremities responded but feebly to the excitation by the galvanic current.

When I examined the patient, on the 12th of December, in Karlsruhe, I was able to corroborate all these statements. As a supplement I may add what follows:

The organ of hearing showed no pathological changes. No alterations were visible; the middle ear was normal, and yet there existed deafness as to sounds of every kind.

When I experimented with Mach's double otoscope and the tuning-fork, I obtained positive and identical results on both sides. The sensibility of the fifth pair was reduced. In the external auditory canal and on the drumhead, pricking with a needle caused the sensation of touch, but no pain. Sensations consequent upon irritation of parts supplied by the fifth pair were but slowly appreciated. Even with 30 Stöhrer's elements, the constant current failed to produce subjective sensations of hearing, though 13 elements sufficed to occasion twitching of the muscles of the face and 24 of the muscles of the extremities. The general motility of the patient was impaired; his movements were but slowly executed, and he became more easily fatigued than formerly. The alteration of the voice was very striking. The patient spoke slowly, and in a whisper; and though he was requested repeatedly in writing to speak loud, he nevertheless continued to whisper. In order to render the diagnosis more complete, we agreed to institute further experiments with regard to general sensibility; and also to examine the urine as to whether it contained sugar.

We were of opinion that an extravasation existed at the origin of both auditory nerves (see our reasons below), and determined to apply a seton in the nape of the neck, to subject the patient to an energetical treatment with iodine, and to make a frequent use of the constant current. This plan of treatment was carried out during the following several weeks.

Upon further examination, no sugar was found in the urine. Anæsthesia not only existed in the district of the fifth pair, but was almost complete over the whole surface of the body. At different parts of the body, deep punctures with a needle caused but a dull sensation.

On the 29th of December, Dr. Picot wrote me as follows: "Examinations made with compasses yield no result on account of the difficulty of communicating with the patient. The surface of the body is everywhere equally insensible to differences of temperature. Ice and very hot water make no impression. The sense of weight is preserved. The patient indicates correctly differences of one pound. Electro-muscular contractility normal; electro-muscular sensibility seems preserved, but reduced. Tickling of the pharynx occasions but tardy and sluggish reflex movements. Currents of the greatest intensity produce no sensation of sound. With the ophthalmoscope no changes found. Urine constantly without sugar. Previous condition unchanged. General health good. Mental faculties intact." Dr. Deimling wrote me that the above-mentioned plan of treatment was followed without altering the condition of the patient, who was discharged from the military hospital and service at the end of January.

The sudden fall was occasioned most likely by a fit of dizziness, the cause of which is not easily determined. Perhaps the tubal catarrh may explain it. It is probable that, in consequence of the fall, an extravasation of blood took place at the origin of both auditory nerves. In no other way can we account for the sudden and total deafness occurring on both sides, and simultaneously with other nervous phenomena. The seat of the extravasation may be placed, with a high degree of probability, in the medulla oblongata at the point of origin of the deep root of the auditory nerve. This root is contiguous to a bundle of fibres constituting in the medulla oblongata the sensitive root of the fifth pair.* According to the researches of Arnold and Deiters, some of these fibres can be traced to the restiform body and the posterior columns of the spinal cord.†

* Prof. F. Arnold had the kindness to show me some very instructive preparations, which leave no doubt as to the close connection between the great root of the fifth pair and the deep root of the acoustic nerve.

† Vide F. ARNOLD—Treatise of Anatomy, Vol. II.; and OTTO DEITERS—Investigations into the Brain and Spinal Marrow. Braunschweig, 1865.

Thus we can explain readily the impairment of sen sibility of the fifth pair. As the disturbances of hearing and sensibility existed on both sides, we may assume that the exudation extended over a considerable area. The pressure of the exudation upon the centripetal and centrifugal nervous fibres, in their course through the medulla oblongata, accounts for the anæsthesia of the whole periphery of the body, and for the reduced energy of motility. The whispering voice may likewise be referred partially to this cause; partially, however, to the circumstance that the patient could not hear his own voice, and therefore did not know what degree of intensity of speech was required in order to be heard and understood as formerly by others.

The lack of sugar in the urine was constant; and we may, therefore, suppose that either none or an insufficient number of the vaso-motor fibres were affected.

It is surprising that even a current of the greatest intensity failed to excite the auditory nerve. In this particular, the case under consideration stands in contrast with a case of recovery from total nervous deafness which I described in vol. I., part II., of these Archives. In this case a current of even low intensity prducoed subjective sensations of hearing. If we consider that, even in some deaf-mutes, the application of the constant current occasions sensations of hearing, which such persons endeavor to imitate, we arrive at the following conclusions:

In cases of considerable impairment of hearing or

total deafness, no prognostic value can be attached to auditory sensations occasioned by the application of the constant current. If, however, a repeatedly applied galvanic current of such intensity as will cause twitching of the muscles of the face and the extremities, fail to produce sensations of hearing, we may infer the existence of complete paralysis of the auditory nerve, and form an unfavorable prognosis.

A CLINICAL ANALYSIS OF THE INFLAMMATORY AFFECTIONS OF THE INNER EAR.

BY H. KNAPP.

THE first incentive to write this paper was received while observing some cases of rapid loss of hearing under such general symptoms as usually accompany the acute perturbations of the circulation of the blood in the brain. As P. Ménière was the first to describe such cases in connection, I began to compare with his statements what had come under my own observation, and to account for the symptoms as rationally as I could. The similarity of appearance which ear diseases of a different nature may present, and the paucity of pathological data, led me to add other observations, the prominent feature of which is a more or less rapid and complete loss of hearing caused by some disturbance of nutrition in the membranous labyrinth. Such cases cannot fail to elicit the interest of the practitioner, as they not only destroy rapidly and unexpectedly one of the principal resources of social intercourse and mental development, but are often ushered in by symptoms we commonly witness in fatal general diseases. My clinical records show that they are by no means so rare as usually represented, and this relative frequency should give them additional claims to the at-

tention of the physician, even if they did not nearly always terminate in incurable deafness. There being hardly another branch of pathology so imperfectly elucidated by post-mortem examinations as the diseases of the labyrinth, I have had to rely chiefly on the records of my own observations, although I have not omitted to draw freely on the literature of the subject. This paper appears, therefore, in the shape of a clinical analysis of a group of the most serious, but also of the least understood, cases that come before the aural practitioner. If their inflammatory nature is not always established by sufficient pathological evidence, they may, at least, be supposed to have their origin in structural changes in the labyrinth. I venture, therefore, to unite them under the common head of inflammatory affections of the inner ear, not being afraid of, but rather soliciting, that men more competent than myself may deem it worth the trouble to set me right where I have erred.

I have grouped the cases partly according to their supposed nature, partly according to their etiology. The former has been done with a few cases only, in order to base the general discussion of our subject on the immediate occurrences of life. The latter has been adopted at the end of the paper, in order to give a comprehensive and practical view of the conditions under which serious labyrinthine affections may be suspected. As the subject is new, but will doubtless soon enlist the attention of observers and investigators, I append to this paper a tabular statement of the literary sources I have had at my command.

Those marked with an asterisk I have read in the original. I, therefore, vouch for the correctness of the quotations made from them: those without asterisks I know from extracts only. The different literary sources are referred to in the context of this paper by the number—in parentheses—which they bear in the appended tabular statement.

Since Dr. P. *Ménière*, of Paris, first described (1) a number of cases of the peculiar disease which is known by his name, only a few confirmations of his observations have been published. Modern text-books do not record anything materially new on the subject, and a tendency seems even to gain ground to explain the striking complex of symptoms known as Menière's disease, by morbid processes in the tympanic cavity acting upon the labyrinth, rather than by a primary lesion of the inner ear, in particular the semicircular canals. *Tröltsch* (2) says: "Manifold demonstrative dissections and various corroborations of the facts relating to this disease are necessary. I myself remember, in my somewhat extensive practice, only a few cases which were analogous to those of Ménière."

The following pages contain what has come under my notice concerning these and kindred affections of the inner ear, during the last twenty-seven months. I shall begin with the consideration of cases of sudden deafness, as representing the purest kind of labyrinthine affections, shall then proceed to cases where the disease of the inner ear is more or less complicated with cerebral symptoms,

and conclude with cases in which the labyrinthine affection is consecutive on diseases of the brain or the constitution. I shall endeavor to analyze these cases in regard to the value of their prominent symptoms, and to determine their nature as accurately as my ability, with the assistance of the literature on the subject, will permit.

CASE I.—*Sudden Deafness Occurring in a Healthy Boy, under the Symptoms of an Apoplectiform Attack.*

The son of F. W. Zothe, of Philadelphia, æt. 19 years, consulted me, September 27th, 1869. He had always been healthy until ten years previously, when, one morning, he was suddenly seized with an apoplectiform attack: his head ached, he was pale, and felt so giddy that he could not stand without assistance. He had nausea, vomited repeatedly, and fainted. All these symptoms succeeded one another in the course of five minutes, remained in their acme for about five minutes more, and then gradually subsided in the manner they had set in, so that the whole attack, from beginning to end, lasted about a quarter of an hour. The recovery was complete, but he noticed at once that he had become very hard of hearing. For a whole year his general health continued undisturbed, but the hardness of hearing remained. Then again, one morning, without any appreciable cause, he had another apoplectiform attack similar to the previous one, which also lasted about a quarter of an hour, and ended in complete recovery, except that his hearing now had become entirely destroyed. He could not hear the loudest sound, not even the whistle of a locomotive. He was treated by the most competent physicians in various ways, but without any result. None of them could discover any abnormality by the physical examination of his hearing organs. At the lapse of about a year a low degree of hearing power returned; he could perceive loud whistling, but no voice. Since that time his hearing has not changed. His health was always good. As his speech grew gradually less distinct, Professor Gross clipped his enlarged tonsils.

When I examined the patient I found his constitution healthy, in the external auditory canals some wax of ordinary quality, both tympanic membranes normal, movable by inflation, and giving the usual sound through the auscultation tube. There was nothing abnormal in the pharyngeal and nasal regions. He could not hear a watch tick, either when it was applied to the auricle or to the cranial bones around. The sound of a tuning-fork was perceived from the forehead and teeth. He could hear loud chords in the three lowest octaves of a piano, but not in the higher octaves. He could hear, indistinctly, loud words shouted in his ears, and was able to repeat their vowel sounds with some correctness. He could speak intelligibly, but had a deficient intonation, and omitted some consonants—for instance, the r.

I considered his affection not amenable to treatment.

This is certainly a most remarkable disease, and so peculiar that it well deserves a special name, taken either from its first scientific describer, *P. Ménière*, or from its prominent symptoms, regarding which the term *Apoplectiform Deafness* seems to be the most appropriate. If the pathological condition, supposed by Ménière, Politzer, and others to be the cause of this striking chain of symptoms were more satisfactorily demonstrated than by the meagre results of only a few autopsies, an appellation based on pathological anatomy would, of course, be preferable, and the disease be called "Apoplexy into the Labyrinth," or "Apoplectic Inflammation of the Labyrinth," in case the ecchymoses be connected with inflammatory changes. Both conditions would be the counterparts of the simple and inflammatory hemorrhages we meet with in the retina and choroid. Unfortunately there is no otoscope to illuminate the labyrinth in the living body. We are, therefore, compelled to form our opinion from the subjective

symptoms, and, in consideration of the scantiness of pathological facts, arrive at the diagnosis of this disease more or less by exclusion.

I will endeavor to appreciate, to the best of my ability, the nature and significance of the symptoms observed in the above described case.

The most prominent feature was the *suddenness of the attack*, almost like a traumatic affection of the ears and brain. There are, indeed, several well-observed cases on record in which an *injury* produced *sudden deafness* under apoplectic symptoms. I may be allowed to give extracts from two of them which are particularly important on account of their having been illustrated by post-mortem autopsies.

A. Politzer (3) reports that a healthy man, æt. 40, who had always enjoyed excellent hearing, experienced a sudden fainting fit, during which he fell with his back on the frozen ground of his court-yard. For several hours he was unconscious. When he awoke, he was completely deaf, unable to speak, and had very severe pain in the back of his head. Soon after he vomited several times. On the next day his power of speech returned, but very loud tinnitus in both ears and vertigo set in, compelling him to remain in bed for four weeks. After that time he could walk about his room, but his gait was unsteady. Though his general health improved, the deafness, tinnitus aurium, and giddiness persisted. Six weeks after the fall he was examined by Politzer; who verified his deafness, but discovered no abnormal condition in the outer and middle ears. At the beginning of the seventh week the symptoms of a severe acute meningitis set in, of which the patient died three days later. Dissection showed a fissure in the base of the occipital bone extending through both the petrous pyramids across the vestibule to the inner wall of the tympanum, which,

however, was not split. The right labyrinth was filled with coagulated blood only slightly changed. Its membranous parts were swelled and softened. The left labyrinth was full of bloody pus, its membranous parts disorganized by purulent inflammation which extended through the fissure into the cranial cavity, causing death by purulent basilar meningitis.

Politzer is of opinion that the fracture of both the petrous bones and the hemorrhage resulting from it caused the symptoms of Ménière's disease, which terminated in death on account of the ensuing purulent inflammation of the labyrinth and base of the brain.

Dr. Voltolini (4) described, in 1869, the case of a soldier who was struck with a piece of wood on the left temple. He instantly fell on the floor unconscious. After some minutes he regained his consciousness, staggered, was put to bed, and vomited. He complained of dizziness and headache, and was completely deaf. There was no bleeding from the ear; no paralysis. Two days later delirium set in, which continued until his death, eleven days after the injury. Two days before death trismus. The autopsy revealed a disastasis of the connections between the parietal, temporal, and sphenoid bones; lepto-meningitis; pus on the base of the brain in the regions of the pons Varolii, medulla oblongata, and cerebellum; the surface of the dura mater over the sella turcica, clivus, and pyramids was likewise covered with pus. A fissure passed through the pyramids of both the petrous bones almost in the same direction, between the fenestra rotunda and the cochlea along the base of the porus acusticus. The left tympanic cavity and the semicircular canals were filled with blood. There was no extravasation in the right tympanum, nor could it clearly be demonstrated in the vestibule or semicircular canals of this ear.

Although the description of this case is less complete than that of Politzer, it shows, nevertheless, the remarkable fact that the symptoms of Ménière's disease were pro-

duced by an unconnected fissure of both the petrous bones, running through the vestibule along the meatus auditorius internus; resulting in hemorrhage in the labyrinth, immediate deafness, and death by subsequent purulent basilar meningitis. This symmetrical fracture in the one case was caused by a fall on the occiput, whereas in the other it followed a blow on the temple.

There are other cases on record in which permanent deafness, accompanied by apoplectic symptoms, resulted from an injury on the head, while all the other symptoms disappeared. In analogy with the two cited cases they may be explained as caused by fissures through the petrous bones with hemorrhage into the labyrinth. Although they terminated in recovery of the patient's general health, the changes in the nervous apparatus of the labyrinth, consequent on the hemorrhage, must have been such as to destroy the hearing function forever. The destructive inflammation, in such cases, should be considered as confined to the inner ear, in a similar way as we find the nutritive changes following embolism of the central retinal artery limited to the retina alone.

Aside from injuries, there are cases of *spontaneous sudden deafness* reported, which were accompanied by one or several symptoms belonging to Ménière's group.

Delstanche (5) relates "that a robust carrier, while walking in the street at the side of his vehicle, was seized with vertigo and fell down upon the ground, without, however, losing his consciousness for a moment. He rose

immediately, but was completely deaf." The result of the examination is not stated.

S. Moos (6 and 7) reports several cases of sudden deafness, "characterized by the absence of any assignable internal or external cause, the negative result of objective examination, the sudden, complete, and permanent loss of hearing, and distinguished essentially from the malady described by Ménière by the absence of the peculiar disturbances of motility, viz.: unsteady posture, staggering gait, rotatory movements, fainting, nausea, and vomiting. The treatment of these cases, also, was nearly always unsuccessful. S. Moos thinks that hemorrhage into the labyrinth most probably is the cause of the sudden loss of hearing in these cases, though they may also be explained in different ways, for instance by paralysis of the acoustic nerves.

Headache may be brought on by the *propagation* of congestion, œdema, purulent and plastic exudation from the middle and inner ear, to the meninges and brain, as we so frequently notice in acute purulent otitis; but it may also be due to simple *irradiation* to the head of the irritation of the sensitive nerves of the hearing apparatus. If the latter assertion do not seem to be borne out by facts, and the objection be raised that we cannot demonstrate it while, on account of the vicinity of the organs, the propagation of congestion would more easily explain the occurrence of headache in ear-disease, I need only remind the reader of the intense headache which accompanies certain forms of entirely localized eye-affections,

especially acute glaucoma. In many cases the headache is so intense, and the other accompanying reflex cerebral symptoms (vomiting, etc.), are so prevalent that the primary—in fact, the only—inflammation of the organ of sight or hearing is overlooked or neglected by the patient and practitioner. Aurists know how many cases of acute otitis, especially in children, are mistaken for meningitis. Such an error is more excusable than when an adult, with acute glaucoma, is treated many a week, as I have seen, for the same disease, although the irritation of the eye and the diminution or loss of sight should have induced both the patient and practitioner to pay more attention to the eye. The cerebral symptoms in acute glaucoma are similar in many points to those in acute otitis media. As glaucoma is an affection limited to the eye, it is certainly justifiable to assume that affections limited to the ear may produce headache and other cerebral symptoms just as well by irradiation of nervous irritation as by extension of morbid products.

Paleness of the face, perspiration, and *fainting,* are the results of disturbances of circulation, temporary anemia. When they occur in ear-diseases, we may consider them as reflex phenomena, and put them in one category with vertigo and vomiting. The reflex action would consist in an irritation of the sympathetic nerve, causing a contraction of the blood-vessels. The connection between the nerves of the inner ear and the sympathetic is unknown. Over-excitement of the acoustic nerves may, either directly, or indirectly through the common sen-

sorium, produce a reflex irritation of the sympathetic nerve, as we see this also after unexpected and vivid impressions of the optic nerves.

Vertigo, a prominent symptom in various severe and sometimes even in slight ear-affections, is generally believed to be the *result of increased intra-auricular pressure.* Its *causes* may be *external:* a plug of cerumen, a foreign body pressing upon the tympanic membrane, exudation and extravasation in the tympanic cavity, the act of syringing an ear when the drum-head is defective, etc.; or they may be *internal:* congestion, exudation, and extravasation within the labyrinth. Further, it frequently accompanies many *cerebral* processes of a congestive and apoplectic character. Vertigo sometimes appears suddenly and violently, and is followed by deafness in connection with other grave symptoms, as in the case first described in this paper, at other times it appears very mildly and temporarily at first, but often returns, leading to gradual deafness with tinnitus aurium and disturbance of general mobility, as in most of the cases described by Ménière himself. Frequently it causes the patient to fall and injure himself. If this injury be but slight and followed by no other symptom than diminution or loss of hearing, as in the case of Delstanche, a *primary* internal ear-disease is diagnosticated; but if the fall has been severe, and followed by cerebral disturbances in addition to deafness, vertigo may have been caused by congestion of the brain, perhaps with consequent pressure upon the labyrinth, but the deafness and other

symptoms may be accounted for more satisfactorily by a traumatic lesion of the cranium with hemorrhage into the inner ear. In this category I may reckon the following observation:

CASE II.—*Sudden Vertigo—Falling down a Staircase—Temporary Unconsciousness—Permanent Tinnitus and Hardness of Hearing—Anomalous Reaction of the Auditory Nerves with the Galvanic Current—Deafness for High Tones.*

Friederich Ege, æt. 48, laborer, of good general health. About twenty-five years ago a gun was fired behind him and a little to his right. He immediately felt a ringing noise in his right ear, which soon disappeared, but the hearing of this ear had ever since been somewhat impaired. His left ear had always been perfect. On the evening of September 12, 1870, in going up-stairs he suddenly felt so giddy that he could not keep his balance, fell backward, slid down to the floor, and was carried to bed unconscious. When he awoke the next morning consciousness had returned. He had pain in his back, great numbness in the right side of his head, and a noise in his right ear like the roaring of a waterfall. His hearing was much impaired in both ears, more, however, in the right than in the left. He left his bed one or two days afterward, but the pain in his back lasted about a month. The noise in his right ear continued unabated for two months, decreased a little during the third, and became more intense again in the last month, when the cold weather set in. A disposition to vertigo was always present. He could never go up or down stairs without feeling dizzy, and was obliged to support himself with both hands on the railing. From time to time he felt a heaviness and numbness in his head. His hearing is somewhat improved.

The patient came to me January 4, 1871. General health good; no disposition to take cold easily; pharyngeal mucous membrane normal. Both tympanic membranes apparently healthy, movable on inflation of the drum, and on suction through Siegle's otoscope. Both Eustachian

tubes pervious. Inflation of the drum producing the ordinary auscultatory sound. He hears a watch of 12′ hearing-distance, at 3″ with the left ear—that is, a hearing acuteness for noises equal to $\frac{1}{48}$ of the normal standard; while the watch is not heard at all with the right ear, either when applied to the auricle or to the temporal or mastoid regions. Ordinary voice is correctly understood at a distance of 20′ with the left ear, and of 2′ with the right. Tuning-forks of different pitch are heard equally well with either ear, both when placed on the glabella and on the incisor teeth. If there is any difference, he can hear them rather better in the right than in the left ear. Sounds on the piano are less distinctly heard with the right ear than with the left, and with the latter less than with the normal ear. The middle octaves are heard most distinctly, the lower less, and the higher octaves least. With the right ear he does not hear the highest octave at all, however loud it may be sounded, while he distinguishes clearly even soft touchings of the notes of medium pitch. He perceives correctly the sounds he can hear, but they seem to come from a greater distance; for instance, when he is near the piano the sounds seem to come from a neighboring house. If the intensity of sound is increased, he thinks that it comes from a less distance, but even the strongest accords on the piano, near which he stands, seem to proceed from an adjoining room through closed doors.

The examination of the patient with the galvanic current, according to Brenner's method, gave the following results:

Jan. 22. Noises in right ear like escaping steam; no noises in left ear.

Right ear: internal arrangement, that is, cathode in ear, anode on back of opposite hand.

With vi elements and 1000 units of resistance: noises ceased after two minutes.

With x el. and 1000 resistances: prickling sensation in ear and hand.

With xv el. and 1000 resistances: distant noises, like the din of the street.

With xxv el. up to 1465 resistances: no reaction.

With xxx el. and 1466 resistances, the following formula:

Ca C,* ringing.

Ca D> (gradually diminishing).

Ca O—

An C—

An D—

An O—.

With xxv el., from 1467 to 1850 resistances, the same formula was obtained.

With xxv el. and 1800 resistances:

Ca C ringing, loud.

Ca D>

Ca O—

An C—

An D—

An O ringing, faint and short.

With xxv el., from 1851 to 2000 resistances, the same formula.

The noises had ceased.

Left ear.—No reaction up to xxv elements.

With xxix el., and without inserting the rheostat, the following reaction was obtained.

Ca C ringing.

Ca D>

Ca O—

An C—

An D—

An O—.

Subsequent examinations showed only slight variations of these results. The quality of the noises changed from a ringing into a singing sensation.

Nausea and vomiting, if not caused by gastric difficulties, are mainly symptoms of cerebral diseases, conges-

*Signification of these Abbreviations.—Ca C=cathode closure; Ca D=cathode duration; Ca O=cathode opening; An C=anode closure; An D=anode duration; An O=anode opening.

tions, inflammations, tumors, etc., in the cranial cavity, but they may also proceed from inflammations of the organs of sight and hearing. Intense iritis, glaucoma, purulent choroiditis, are usually accompanied by sickness and vomiting. Every ophthalmic surgeon knows the dismal prognostic significance of these symptoms when they occur soon after an operation. That they frequently accompany the acute and severe, especially purulent forms of otitis media, is well known to all aurists. Troeltsch (2) and others point out correctly that vomiting is sometimes also observed in *milder catarrhal forms of otitis media.* A very instructive case confirming this statement has been published by J. Orne Greene, of Boston (8).

A carpenter suffered for a year from a simple chronic catarrh of one ear, with severe vertigo and vomiting, which mostly occurred in the morning, and frequently compelled him to interrupt his work. All internal remedies proved unsuccessful, but inflations of air into the tympanum, and injection of a solution of alum (gr. x ad ℥i aq.) through the Eustachian catheter, cured, in the course of a few weeks, the aural catarrh, with which also the vertigo and vomiting disappeared.

This case demonstrates that the causes of vertigo and vomiting may be sustained even by *chronic* inflammations in the tympanic cavity, and, on account of this fact, Greene's observation, showing that these aggravating symptoms were removed by the ordinary treatment of aural catarrh, is of great importance.

Regarding the course of this case, I am allowed to add the following statement received, by letter, from the author:

"More than a year afterwards, the man came to me again, with a slight return of the vertigo only, having remained well all that time. The hearing was about the same as when he ceased treatment. Inflation of the ear once relieved him at the time, and I have heard nothing more from him, as I think I should have done had he been troubled again."

Vomiting is a common symptom in many diseases of the various organs of the body. In inflammation of the ear no significance can be attached to it other than that from the acoustic or sensitive nerves of the organ of hearing, by the way of reflex action, some irritation of the centre of the pneumogastric nerve is produced. This opinion is based on the physiological fact that, after the division of the pneumogastric nerve in animals, excitation of its *central* end produces vomiting. Whether an irritation may be reflected directly, viz., without the interposition of the pneumogastric, from the nerves of the ear to those regulating the peristaltic motion of the stomach, is unknown, but seems not impossible.

Tinnitus aurium, although not noticed—perhaps only not remembered—in the first case here described, is a nearly constant symptom in Ménière's disease. Subjective sounds and noises may be considered always to result from irritation of the nervous apparatus of hearing. They will, therefore, be met with in all diseases in which the auditory nerve, and especially its final expansion in the labyrinth, suffer from pressure, inflammatory changes, or hyperæsthesia. The ear diseases in which it occurs are, therefore, even more numerous than those which cause vertigo.

The most general symptom of ear disease is *impairment of hearing.* All the changes in the external and middle ear which constitute an obstacle to the transmission of sound to the labyrinth, all affections of the roots, the course, and the termination of the acoustic nerves, which are not of a purely irritative nature and cause hyperacusis and tinnitus, will produce impairment of hearing.

All the symptoms enumerated thus far are less characteristic of a labyrinthine affection than the *disturbances of equilibrium : uncertain posture, staggering gait, and rotatory movements,* which complete the chain of symptoms comprehended under the term of Ménière's disease, or apoplectiform deafness. As these symptoms have not been noted in the cases before described, I will here report another one in which they were well pronounced.

CASE III.—*Sudden Appearance of Headache, Cloudiness before the Eyes, Nausea, Retching, Tinnitus, Impairment of Hearing, Vertigo, Unsteady Gait, Turning to the Right Side, Vomiting, Recovery from General Symptoms, Impairment of Hearing Remaining, Deafness for High Tones.*

Frederick Kuhn, smith, aged 46, of New York, suffered for the last three years with shooting pain in his legs. The pain occurred in attacks which began in the evening, lasted uninterruptedly from 12 to 48 hours so as to deprive him of sleep, then ceased completely and reappeared usually after three or four days. During the last year these attacks gradually became less intense, lasted a shorter time, and disappeared in the beginning of January, 1871. His general health was not impaired.

On arising one morning, at the commencement of November, 1870, he experienced frontal pain, heaviness of the head, cloudiness before his eyes, nausea, efforts to vomit, ringing noises in both ears, and impaired hearing, obliging him to ask people to speak louder. He felt *giddy;* objects around seemed to move; he was *unable to stand firmly*, and when trying to walk, he *staggered* and *turned involuntarily to the right side.* The giddiness diminished when he lay down.

These symptoms lasted unabated for one hour, and then gradually subsided in the course of half an hour; the noises in his ears and the hardness of hearing only remained. He also felt a certain weakness in his legs. Toward the evening of the same day he vomited several times, which recurred an hour after dinner on the three or four following days. The vomiting was unaccompanied by giddiness or headache. He has had no other similar attack, but his hearing has remained impaired, the noises in his ears have persisted, and his gait has been uncertain ever since. He has also observed that his sight was failing.

He came to the New York Ophthalmic and Aural Institute February 22d, 1871. The examination with the galvanic current gave the following result:

Right ear. xx el. and 2000 resistances:

Ca C loud knocking.

Ca D >

Ca O—

An Cl loud knocking.

An D >

An O—.

Left ear the same, and loud knocking sensation on An O.

February 17th, 1871, I made another examination and found the following conditions: The patient looked healthy, and complained only of the failing of his sight and hearing, in addition to a weakness in his legs.

Acuity of vision $\frac{2}{200}$ in either eye, but increased to $\frac{2}{40}$ by convex glasses of 26″ focal distance. With + 10 he read finest print with some difficulty. The visual field of either eye was much contracted,

viz., to about 9″ in diameter in the left eye and 6″ in the right, the examination being made with the hand moved 1′ before the eye. The contraction was excentrical, with the point of fixation near its inner border. Perception of colors perfect. Both optic discs very white, vessels scarce, arteries smaller, the remainder of the fundus normal.

Watch heard when applied to the auricle, but not from the temporal or mastoid region. Ordinary voice R. 15′, L. 5′. He perceived correctly and acutely all the sounds of a piano *except the highest.* With the right, his better ear, b''', and with his left ear d'''' were the highest notes he perceived. The limit was abrupt for the right ear, so that c'''' and any note above it sounded even much louder than b''', were not perceived; in the left ear the tones began to fade away from b''' upward, but he distinguished them so far as e'''', when I proceeded to sound them from below upward, but he began to hear them only at c'''' or d'''' when I sounded them from above downward. A low tuning-fork was heard from all parts of the skull. Right tympanic membrane somewhat white, and a vessel visible along the manubrium; left tympanic membrane entirely normal; both easily movable on suction and inflation. Valsalva's experiment positive on either side. No catarrh of fauces, pharynx, or larynx, nor has he ever had a tendency to such affection. His gait has become more steady and certain, although he has to look attentively at the floor, on account of the restriction of his visual field. All the other symptoms of the attack have likewise left him, except his diminution of sight and hearing, and a slight ringing noise in his ears.

This case is a fair specimen of Ménière's disease. It began with a sudden attack of the cerebral symptoms, which we have observed in the first case, and discussed. It differed from the first case by the longer duration—one hour and a half—of the attack, the lower degree of the impairment of hearing, the addition of tinnitus aurium, distinct disturbances of equilibrium, and symptoms on the part of the organs of sight.

The lower degrees of disturbance of equilibrium, manifesting themselves in the uncertainty of station and gait, may be dependent on a vertiginous and fainting condition, not in itself indicative of a labyrinthine affection, when they are observed during the attack. In fact, nobody, during an attack of pure apoplectiform congestion of the brain will be able to stand and walk with his usual certainty, if he be able to stand or walk at all. But when these symptoms, together with tinnitus aurium and impairment of hearing, persist long after the attack has ceased, they not only strengthen the diagnosis of a labyrinthic affection, but even localize it—denoting that the *semicircular* canals are implicated in the morbid process. Since the celebrated experiments of Flourens (9), which showed that the lesions of the semicircular canals were followed by disturbances of equilibrium and motion, many physiologists (10, 11, 12, 13, 14, 15) and physicians (16) have made special investigations into this subject, which, with one exception (Schiff), have corroborated the *results obtained by Flourens*. These were, in brief, the following: If the horizontal semicircular canal in one or both ears is divided, the head, and frequently the whole body of the animal perform rotary movements from right to left, or the inverse. If one vertical canal on each side is severed, the animal moves its head constantly up and down, and has a tendency to fall forward or backward. If several canals are divided, movements resulting from the lesion of the injured canals combinedly follow. The movements are inordinate, as if the animals suffered from

vertigo. If only the osseous, and not the membranous semicircular canals were divided, these abnormal movements did not occur. If the semicircular canals were not divided, but simply to some extent destroyed, the animals lost their equilibrium altogether, and perished soon by wild whirling motions and falling forward and backward. If only the semicircular canals were injured, the animals did not lose their hearing, and if the cochleæ alone were injured, the animals lost their hearing, but showed no disturbances of equilibrium and motion. Czermak (11) found that pigeons, after injury of the semicircular canals, frequently have vomiting. Goltz confirmed that the animals thus injured have vertigo, and dwells on a rational explanation of these remarkable phenomena. If the auditory nerve and its expansion in the labyrinth, he argued, were merely subservient to the sense of hearing, this could not satisfactorily account for the surprising disturbances of motility. He arrives at the conclusion that the two fascicles of the auditory nerve have different functions: the cochlear fascicle is the nerve of the special sense of hearing, but it is questionable whether the semicircular canals are also organs of audition. Being subservient to the maintenance of poise, *they are*, as it were, *the organ of the sense of equilibrium of the head, and thereby of the whole body.* Goltz gives a highly interesting analysis of this theory, explaining how he imagines the maintenance of equilibrium to be effected by the semicircular canals. The terminations of the nerves in the ampullæ and canals

are excited by pressure or tension like the tactile nerves of the skin. The liquid in the semicircular canals—endolymph—according to physical laws, distends most those portions of the wall which lie deepest. The pressure from the fluid varies with the movements of the head, so that a determined nervous excitement corresponds to every position of the head. The perception and consciousness of this special nervous excitement by the brain constitute the sense of equilibrium, which serves as a regulator of the movements. If a portion of the semicircular canals is injured, the brain receives inaccurate information of the position of the head, and is unable to calculate and direct its movements correctly. This is the cause of the vertigo and the disturbance of motility.

If a lesion of the semicircular canals exists on one side only the disturbances are temporary, but if it exists on both sides they are permanent. Goltz says that this is no objection to his theory, as also the loss of one eye does not produce blindness; the organ of *one* side may soon be sufficient to inform the brain of the position of the head. Goltz shows further that the movements of the body essentially depend on the position and movements of the head. Disturbances of the latter are *immediate* consequences of the lesion of the semicircular canals, while those of the equilibrium and general movements of the body are *secondary*.

If we now return to the analysis of our case (the third), we note that the patient "felt giddy, the objects around him seemed to move, he was unable to stand firmly, stag-

gered when trying to walk, and turned involuntarily to the right side." To suppose a lesion of the semicircular canals as the cause of these symptoms would be in perfect accordance with the physiological statements just described. The giddiness—the loss of the sense of equilibrium—diminished when the patient lay down, which is in harmony with the fact that giddiness from any cause always diminishes when part of our body finds a support which, by the aid of the nerves of touch, aids us to become better cognizant of the position or movements of our body in regard to the surrounding objects. The vertigo was the immediate consequence of the lesion of the semicircular canals; the disturbance of equilibrium of the body, the secondary consequence. The unsteady standing, the staggering gait, are only indetermined signs of the loss of equilibrium; but the tendency to turn to the right side indicates that the horizontal canals were principally affected, and the vertical comparatively uninjured, as the patient had no tendency to fall forward or backward. The cochlear portion of the labyrinth must also have been affected, as the patient had become hard of hearing. The diminution of hearing was greatest in the left ear, a proof that the cochlea of this ear was more seriously injured than that of the right. Are we allowed to assume that the semicircular canals of this ear were also more affected than those of the better ear, in order to draw some conclusion concerning the disturbances of motility? It would seem natural to assume that the canals were more injured in the worst ear. As our patient had a

tendency to turn to the right side, the conclusion would be that the rotatory movements are executed from the side of the affected ear to that of the good, respectively better ear. This conclusion, however, would be at variance with other observations. Trousseau (16, p. 550) reports a case of Ménière's disease. The patient "felt herself pushed from left to right; when walking on the pavement she was always careful to take the right, being afraid of falling upon the causeway. The deafness was greatest on the right side, and noise produced a painful impression on the same side." Trousseau adds: "It is now generally admitted by physiologists, that the simultaneous lesion of the semicircular canals on both sides gives rise to forward movement or backward movement (a tumble head-over-heels), according to the situation of the injured canals; and that when there is lesion of one side only, the propulsion is always to that side." In confirmation of this the celebrated case of MM. Signol and Vulpian's rooster is quoted. "A cock received a violent stroke on the head from the beak of another cock. The animal was at once stunned, but was soon seen, with head drooping forward, to turn himself *from left to right* when attempting to walk. He became blind. When he died, six weeks after receiving the wound, a great part of the right temporal bone was found to be necrosed: the whole of the portion of the bone containing the semicircular canals was isolated by a newly formed membrane, and it was impossible to discover any trace of the semicircular canals of the right side."

This case proves unmistakably that the rotatory movements are from the healthy to the diseased side. The clinical observations are not quite conclusive. It is possible that the cochlear portion of the auditory nerve is more diseased in the one ear, and the vestibular portion more in the other. The patient will then feel an impulse to turn to the side of the better hearing ear, as in our third case. But Trousseau himself (16, p. 553) quotes the case of Dr. Hillairet (17), in which all the symptoms of Ménière's disease were produced by purulent otitis media and formation of polypoid granulations. "There was buzzing in the affected ear, vertigo, a tendency to vomit, fidgets, feebleness of the inferior extremities, a disposition to stoop, and to turn to the side *opposite* to that which was the seat of the lesion."

Such contradictory statements show that further physiological, clinical, and pathological researches are needed to establish, in detail, the dependence of the direction of the rotatory movements on the lesion of the semicircular canals. It does not seem demonstrated beyond a doubt whether the lesion of the *horizontal* canals is sufficient to produce the rotatory movements, nor whether the body turns always to the side of the affected ear.

Before we proceed further, it will not, perhaps, be inopportune to present an abstract of the publications of *P. Ménière* himself (1), on account of their great importance in the description of the disease which bears his name; all the more so as they are not easily accessible, and as a complete synopsis of his cases has never been made.

Synopsis of Ménière's Cases.

Case I.—(*Observation I.*, l. c. pp. 88—89, continued on p. 239.) A doctor of medicine, æt. 47, had for twenty years *tinnitus;* gradual *impairment of hearing;* no appreciable affection of middle ear on either side, but frequently congestion and swelling of outer ears. One day while reading he was suddenly seized with violent sneezing. On arising he was unable to move in a straight line, and his *gait* was *staggering.* Three hours later the same symptoms returned. He went to bed and slept several hours. When he awoke he found that he walked with more difficulty than the day before, had the sensation of heaviness and compression in his left occipito-mastoid region, and his body turned involuntarily from the right to the left side. He was in danger of falling down, as if his left side no longer obeyed his volition, while, when lying in bed, he could move both sides at will. His mind was clear. Soon after he felt sick, and vomited repeatedly. In the course of five days, under the influence of a venesection, cups, and a purgative, his difficulty of motion gradually disappeared; he could walk out again, not, however, without some uncertainty. Forty-two days subsequently, sudden indisposition, and vertigo which the open air did not dispel. He vomited, walked very staggeringly, had *convulsions of his face*, the *left side* of his body was singularly *contorted and soon paralyzed*, but incompletely. These symptoms continued for some days, disappeared under mild aperient medication, but left his hearing weaker than it had been before.

Case II.—(l. c. p. 89.) M. H., M.D., æt. 45, after intermittent fever, had constant *tinnitus* aurium, itching in and slight discharge from the external auditory canals. Soon afterward *sudden vertigo*, nausea, and *vomiting*. *Several similar attacks* in the course of the year, during the last of which the giddiness was so severe that he suddenly *fell down* while walking in the street.

The *hardness of hearing* rapidly increased, and two years afterwards he was *almost deaf*, while his cerebral functions remained undisturbed, and his general health was completely restored. Inflation of the tympanic cavities was easy, *the ears*, with the exception of some slight discharge from the external meatus, being *healthy*.

Case III.—(*Observation II.*, p. 239.) A young, healthy man *suddenly fell down*, as if struck by lightning. He was raised, his members being inert, his *face pale*, bathed with perspiration. *Nausea* and *vomiting* followed. After a momentary loss of consciousness, every-

thing around him seemed to turn. He experienced intense *tinnitus* aurium, and was *hard of hearing*. Before the attack his ears and hearing had been normal. The recovery of his general health, under severe antiphlogistic treatment, was slow. Afterward he had *several similar* but less violent *attacks*, from which he quickly recovered. His hearing, however, diminished gradually, and he heard constant noises. It was *difficult* for him to *keep his equilibrium*, and he could not turn round rapidly without feeling some vertigo. On physical examination, the outer and *middle ears* were found *healthy*.

Case IV.—(*Observation III.*, l. c. p. 240.) A stout merchant, 40 years old, *fell suddenly*, while sitting in an arm-chair, with the upper part of his body on the left arm of the chair. He was *unconscious*, his *face* was *pale* and bathed with perspiration. When raised, he fell to the left side again. *Nausea* and *vomiting* set in. Being put to bed, he regained his consciousness, but felt very ill. Everything about him seemed to *turn*, as if he were sea-sick. He took hold of his bedstead for fear of being thrown over. The vomiting continued. Under antiphlogistic treatment he was gradually relieved, could stand again, but had a slight sensation of uncertainty in walking, violent and constant *noises* in his ears, and *impairment of hearing* which gradually increased. Before the attack his hearing organs had been perfect.

Case V.—(*Observation IV.*, l. c. pp. 240 and 241.) A very nervous young man, from the south of France, had had *repeated attacks of sudden giddiness, nausea, and vomiting*. He had been treated for cerebral congestion with venesections, leeches, and purgatives, but on noticing that his *hearing* had become *impaired*, that *tinnitus* aurium had set in, and his *gait* become *staggering*, he went to Paris. Ménière found the external and middle ears healthy. The patient had a similar attack in Paris, which lasted only a few minutes. As nothing abnormal in the cerebral functions could be detected by Trousseau and Ménière, an alteration of the inner ear was assumed to be the cause of the above-mentioned symptoms.

Case VI.—(l. c. p. 379.) A healthy man had frequent attacks of *vertigo*, *fell down suddenly*, lost his consciousness, sensibility, and power of motion. He always recovered in a few minutes, but *constant noises* and *impairment of hearing* appeared, which increased gradually. These periodic attacks persisted for more than a year, then ceased, and left no other disturbance than complete deafness. Fifteen years afterward the patient still enjoyed excellent health, but remained deaf.

Case VII.—(l. c. p. 379.) A young lady *felt suddenly* a "blood-

stroke" (*coup de sang*) in her left ear, together with a *fainting* turn, *giddiness*, and *noises* in her ear. She went to bed and passed a rather uncomfortable night. The following night she was *suddenly seized* with violent and persistent *vomiting*. She could not sit in her bed without feeling as if she were in a ship on a stormy ocean. She noticed that she could *hear* almost nothing *with her left ear*. The cerebral symptoms disappeared gradually in the course of two months, but the *uncertainty in walking*, a tendency to *vertigo*, and the *deafness* of her left ear remained, while the right was healthy. No lesion could be detected in either ear. All remedies, the electro-magnetic current included, were inefficacious.

CASE VIII.—(l. c. p 379.) A robust farmer, æt. 20, was *suddenly* seized with slight *vertigo*, *obscuration of his visual field*, and *nausea*. He was obliged to sit down, and sometimes *fell* on the floor, with *momentary loss of consciousness*. These attacks recurred more than twenty times in the course of a year, and were never accompanied by convulsions or vomiting. After this year there were no such attacks, but there remained a diminution of hearing, especially in the left ear. At the end of twelve years hearing was destroyed on the left side, and so feeble on the right that ordinary conversation was maintained with difficulty. Although otherwise perfectly healthy, and having never suffered any inflammatory affection of the ears, his deafness annoyed him so much that he went to Paris to seek relief.

Dr. Ménière could detect no appreciable lesion in any of the accessible parts of the organs of hearing. The left ear was totally deaf; but with the right he could hear a loud and distinct voice at a short distance. His parents were healthy and had no defect of hearing, but a sister, then 24 years old, had become gradually deaf without any of his syncopal attacks or any appreciable disease of the ears.

CASE IX.—(l. c. pp. 379, 380.) A healthy coachman of good habits, 38 years of age, suddenly observed *disturbance of vision*, *cloudiness* of the visual field, and a sensation of *wavering* of objects. He soon felt *vertigo*, but *no tinnitus*. After a bad night, on trying to rise he could not hold himself erect, but was obliged to lie down again. When he tried to sit up he felt as if everything *turned* around him. These vertiginous attacks were produced by every attempt to sit up during a period of two months. *Tinnitus* aurium appeared at the commencement of the disorder, and a progressive *diminution of hearing* in the left ear was observed after the third week. When he came to Paris he could walk, but

had *lost* the sense of *equilibrium*, especially on the left side. His gait was unsteady, and he had no confidence in his sight.

Dr. Ménière found the hearing apparatus on both sides physically perfect, and hearing on the right side good, but entirely lost on the left.

Case X.—(l. c. p. 598.) A young woman, menstruating, took cold while riding on the top of a stage coach and *suddenly became deaf*. She was received into the wards of M. Chomel. Her principal symptoms were persistent *vertigo* and frequent *vomiting*. She died on the fifth day. The *autopsy* showed the *cerebrum, cerebellum, and spinal marrow perfectly healthy*. Dr. Ménière examined the auditory apparatus on both sides, and found *alterations only in the semicircular canals*, which, on both sides, were filled with a reddish plastic substance—a kind of bloody exudation—replacing the liquid of Cotugno. Traces of this exudation were present in the vestibules, but the cochleæ were normal.

A review of these cases shows a chain of symptoms which have been already discussed. More or less sudden attacks of grave cerebral disturbances—headache, vertigo, fainting, nausea, vomiting—accompanied and followed by tinnitus aurium, impairment of hearing, and loss of equilibrium. The latter consisted in all cases in the inability to stand and walk firmly, the patients actually falling down or being prevented from falling only by the by-standers. Falling may, according to physiological deductions, indicate that the vertical semicircular canals are affected, but it may as well, and more simply, be considered the consequence of the vertiginous and syncopal condition of the patient, as we frequently see in cases where no lesion of any part of the auditory apparatus can be suspected. In eight of the ten cases described by Ménière, no turning motions are mentioned. Disturbances of equilibrium, therefore, are always present in the disease under consideration, but they have only rarely a

well-pronounced rotatory character. Prof. Goltz's experiments and conclusions explain this easily enough: If the horizontal canals only are implicated, the rotatory movements are conspicuous; if the vertical or all the canals are affected, the disturbances of motion are complex, manifesting themselves principally by the inability to stand and walk with any degree of certainty.

The symptoms I have thus far described may be considered the *more characteristic symptoms* of Ménière's disease. Among the accessory ones I may mention the following:

Loss of consciousness, observed sometimes during the attack, more frequently altogether wanting or very transitory, and to be considered dependent on a syncopal condition.

Cloudiness of the visual field is sometimes (in 2 out of the 10 cases of Ménière) observed during the attack, but does not last long or lead to permanent diminution of vision. I do not think that it results from any abnormal condition, such as hyperemia of the choroid, with effusion into the retina and vitreous body, but rather from the anemic condition of the brain and retina connected with the syncopal state during the attack. The permanent asthenopia and amblyopia in our third case had, on closer examination of the visual organ, to be attributed partly to causes unconnected with the severe cerebral symptoms; they may have had, however, also in part a cerebral origin. The asthenopia was due to an anomaly of refraction of the eye, hyperopia, and

was removed, together with the greater part of the apparent amblyopia, by the aid of proper convex spectacles. The remaining minor part of the amblyopia, however, was due to a low degree of atrophy of the optic nerves, for which no cause could be detected within the globe, and which, therefore, must be designated as extra-ocular, that is cerebral or spinal. The peculiar lesion of the brain, which in this and many similar cases determines the atrophy of the optic nerves, cannot, as yet, be defined. I deem its simultaneous occurrence with nervous ear-disease worthy of notice, and would direct the attention of aurists to the importance of examining the condition of the eye, and that of the optic nerve in particular, whenever a case of nervous deafness comes under their observation. Such examinations are apt to facilitate the diagnosis, and may throw some light on the localization of the morbid processes causing the so-called nervous deafness. It would be desirable to know, approximatively at least, the percentage of the simultaneous occurrence of nervous deafness and nervous blindness.

A peculiar symptom which I observed in the second, third, and forty-second cases, and do not find mentioned anywhere in connection with Ménière's disease, is deafness *for certain groups of tones.* In the second case it is stated "that the patient heard the middle octaves of a piano most distinctly, *the lower less* and *the higher octaves least.* With the right ear he does not hear the highest octave (g''' to g'''') at all, however loudly it may be sounded, while he distinguishes clearly even soft touch-

ings of notes of medium pitch." In the third case the patient could hear correctly all the sounds of a piano, *except the highest*, from b‴ with the one (the better) ear, and from about d⁗ upward with the other. The limit was higher when the pitch of the tones sounded on the piano was successively increased; the limit was lower—that is, the defect greater—when the pitch of the tones was successively diminished. This is an observation common to all sensuous perception. It is generally known that a watch ceases to be heard at a greater distance from the ear when gradually removed from it than it begins to be heard when approximated to it. The visual field is larger when the visual object employed in measuring it is gradually removed from the eye than when, from beyond the limits of the visual field, it is approached to it. The forty-second case, Ménière's disease resulting from syphilis, exhibits the same conditions. It is reported near the end of this paper. In some other cases, for instance the fourth, the vibrations of high and low tuning forks were not heard, but those of tuning forks of medium pitch were well heard.

DEAFNESS FOR CERTAIN GROUPS OF MUSICAL SOUNDS IS PROOF POSITIVE THAT THE DISEASE IS NEITHER LIMITED TO THE ACOUSTIC NERVE OUTSIDE THE LABYRINTH, NOR TO THE SEMICIRCULAR CANALS AND VESTIBULE, BUT THAT IT EXTENDS TO THE COCHLEA. If there existed only a general diminution of the hearing acuteness for musical sounds, a pressure upon, or atrophy or some other change in the nerve itself, or in the centre of audition, might be

assumed, and this supposition would not even be entirely indefensible if only one part of the musical scale, for instance the highest or the lowest notes were not perceived, for we may justify the assumption that a tumor pressed only on one side of the acoustic nerve and destroyed or suspended the conductibility of the corresponding fibres. Our third case would be open to this objection. In the second case, however, we find both the (opposite) ends of the musical scale lost, and it would be absurd to assume that a tumor or some other morbid product pressed simultaneously on two opposite sides of the acoustic nerve and left the centre intact. It would be not less absurd to assume that some pathological process had acted upon two opposite portions of the centre of audition in the brain, and left the intermediate portions intact. This interesting observation of deafness for the highest and lowest notes of the piano can only be accounted for by attributing it to some change in that portion of the labyrinth which encloses the expansion of the cochlear nerve. It is *a contraction of the range or field of audition*, as we have contractions of the field of vision. The latter is observed in different eye-diseases, but the kind found in *glaucoma* most resembles the contraction of the range of audition. In glaucoma we notice first a defect in the innermost portions of the visual field, which extends gradually to the upper, lower, and outer portions. In our third case we noticed deafness for the highest notes alone; in the second we found the same more extended and combined with hardness of

hearing for the lowest tones of the piano. This second case seems to be in a farther advanced stage than the third. I need not point out how desirable further clinical study of these conditions is. I refrain from making any comparison between glaucoma and Ménière's disease. If increase of tension can be demonstrated as a fundamental symptom in the latter difficulty, further analogous conditions will not long fail to be detected.

Having now described and analyzed the symptoms of the disease, we may ask, *Where is its seat?* The pathological anatomy of this affection is still very fragmentary. The two above described cases of traumatic labyrinthine deafness (Politzer's and Voltolini's cases) show purulent inflammation and hemorrhage in the different parts of the labyrinth. Ménière's tenth case reveals hemorrhagic exudation in the semicircular canals only, extending in a minor degree into the vestibule, but not at all into the cochlea. In this latter case the patient was so much prostrated that her hearing power was not tested which would have been of great importance, as the cochlea contains the expansion of the proper nerve of audition.

This *scarcity of pathological* facts compels us to appreciate the more the results of *physiological* research, and therefore I have entered with so much detail into the analysis of the symptoms of the disease under consideration. That the semicircular canals are implicated is evident by the disturbances of equilibrium more or less pronounced, but present in all cases. Enough has been said on that point. Ménière and many authors seem to restrict

the derangements of structure to the semicircular canals and the vestibule. This appears improbable when we remember that diminution of hearing in general, not only of noises, is one of the essential symptoms of the disease. From this it follows that either the trunk of the acoustic nerve, or its final expansions in the labyrinth, or the centre of audition in the brain, must be altered. Our observations on the deafness for high and low musical sounds, a contraction of the range of audition, decide this question beyond a doubt, and localize the derangement within the cochlear canal.

Thus, *anatomical, and principally physiological facts, prove unmistakably that in Ménière's disease we have to deal with true nervous deafness, the different portions of the labyrinth being involved.*

But now another question of the greatest practical importance arises, *Is the labyrinth in Ménière's disease primarily affected, or only influenced from the adjoining tissues in the tympanal and cranial cavities?* A priori, we should suppose, according to the principles of general pathology, that there must exist, as in almost all organs, a primary and a secondary, or, in other words, an idiopathic and a consecutive inflammation of the inner ear. And if the difficulty consists in, or, at least, begins with hemorrhage into the labyrinth, as we may infer from the very acute, almost sudden appearance of the attack in some cases, for instance our first, then the question is: Was the hemorrhage not favored by certain pre-existing morbid conditions in the brain or tympanum?

This question we may certainly answer in the negative for some cases. If a young man never had an aural or cerebral trouble of any kind, and is certainly seized with headache, vertigo, vomiting, and loss of hearing, and if all the general symptoms disappear completely in the course of a quarter of an hour, leaving only deafness and tinnitus aurium, we should be hypercritical in diagnosticating anything but primary labyrinthine affection, probably of an apoplectic nature. The same may be asserted of other cases, for instance our third, and most of Ménière's cases, where the first attacks were also more or less sudden but protracted, and relapsing from time to time, not showing, however, any evidences of tympanal or cerebral affections. I should not omit to repeat that the low degree of atrophy of the optic nerves in our third case argues in favor of some cerebral disorder. The sudden appearance of the aural difficulty, its typical development and course, and the absence of any other cerebral derangement, determine the case likewise to be an example of idiopathic labyrinthine affection, accidentally complicated with a low degree of atrophy of the optic nerves.

Active morbid processes in the middle ear, acute catarrhal and purulent (phlegmonous) inflammations, may cause severe cerebral symptoms, either with or without implication of the labyrinth. Lucae (18) points out that the labyrinth may be assumed uninvolved, if the sound of a tuning-fork from the glabella is heard better, or at least equally well, on the affected side. The prognosis quoad vitam, he says, is favorable in such cases.

Although this may not hold good for all cases, as the inflammation frequently spreads from the tympanum into the brain through the tegmen tympani directly, it is certainly a valuable diagnostic symptom of the fact—which is also corroborated by pathological anatomy—that the labyrinth may be uninvolved in severe affections of the middle ear. On the other hand, the labyrinth, beyond a doubt, in many cases of tympanic affections, participates more or less extensively. Hyperæmia with serous and purulent effusions have been found in all parts of the labyrinth. Certain functional disturbances, such as tinnitus aurium, diplacusis monauricularis and binauricularis, and deafness for sounds transmitted through the bones, also indicate a participation of the labyrinth when occurring with morbid conditions of the middle ear. The diagnosis and denomination of the disease in such cases will hardly be difficult, although it may often not be easy to determine to what extent the labyrinth is involved, and what is the anatomical nature of the secondary changes. Tinnitus, diplacusis, and paracusis may result from increased intra-auricular tension, which, of late, has been called in to explain so many symptoms not otherwise explicable. Chronic otitis media, however, may lead to the production of Ménière's disease. In some of the cases published by Ménière himself, this origin, if not clearly expressed, may be supposed with probability. It can easily be understood that chronic inflammation of the middle ear produces a hyperæmic condition of the inner ear, which may persist after the aural catarrh has ceased.

This labyrinthine congestion, although resulting from catarrh in the tympanum, may itself be the origin of the lesion causing the chain of symptoms present in Ménière's disease. The following case affords an illustration of this proposition:—

Case IV.—H. Barry, of New York, 32 years old, has always enjoyed good health and excellent hearing until 3½ years ago. In August, 1867, he experienced, after bathing in the sea, impairment of hearing and noises in both ears, without pain or discharge. Both the noises and impairment of hearing have remained permanent, increasing in bad weather. He was treated in different ways by aurists of this city and of Paris. In November, 1869, when he first came to consult me, I found all the symptoms of chronic aural catarrh present in both ears. The tympanic membranes were dull, movable, and slightly depressed. The ticking of a watch was not heard at all, the tuning-fork from the glabella was well perceived, loud voice was heard at 5 feet with the right ear, and at 10 with the left. He suffered from chronic pharyngo-nasal catarrh, and constant tinnitus aurium. Inflation, according to Politzer's method, produced a slight crackling sound, heard through the auscultation tube. The tick of the watch was afterward heard ½ an inch from the right ear, and on contact with the left; the voice some feet farther with either ear than before. I treated him with astringent remedies applied as gargles, injected with the posterior nares syringe, and snuffed up through the nostrils, together with inflation of the tympanic cavities. He improved so much as to hear the voice at 12 feet with the right ear, and at 20 feet with the left, and the tick of the watch at 1 inch with the right, and ¼ inch with the left ear. The noises had almost disappeared after he had followed this treatment pretty regularly for four months. During the last year he came to see me only at long intervals, when a cold had produced a temporary aggravation. In the beginning of March, 1871, he saw me again, complaining that during a long spell of stormy weather he had taken a fresh cold by getting his feet wet. I found his hearing diminished in either ear to the perception of ordinary voice at the distance

of half a foot. He could not hear the watch at all, and said that the noises in his ears were intense. After inflation of the tympanum, he heard the voice at 2 feet with each ear. I injected a 1 per cent. solution of nitrate of silver into his pharyngo-nasal cavity, and advised him to apply a vesicant behind his ears. He felt better for two days, and slept well; but when he awoke in the morning of the third day he had headache, vertigo, and nausea. On raising his head, his bed and the surrounding objects seemed to tilt. The pictures on the wall appeared to be in a see-saw motion. He felt as if he were on board ship upon the ocean in a storm. When he sat up, his head moved involuntarily from one side to the other, as if irresistibly pushed; at times it also was forcibly drawn backward, but never forward. By great effort he got up, and drove in a carriage to his place of business, but was obliged to return at once, as the above-described symptoms became so intense that he was unable to keep himself upright. In standing or walking he reeled from side to side, and prevented himself from falling by grasping surrounding objects. His hearing was worse than before, and the noises in his ears very distressing. In his right ear they resembled a sharp whistling, while a humming, like that of insects, seemed to fill the whole left side of his head. His own voice sounded to him shrill and whistling. While lying quiet in bed he felt easy, but the moment he tried to rise the vertigo and turning motions reappeared. These symptoms were less marked the next day, and he was able to go out the third day; but his gait was so unsteady that he frequently was obliged to take hold of a lamp-post or railing for fear of falling. The inclination to fall was always sideways, never forward. During the following two weeks he improved gradually, the symptoms returning in rarer and shorter attacks. On interrogation he remembered having had previously such slight attacks, but as they were only momentary he had never mentioned them. He told me the history of the severe attack and its course, on March 23, a fortnight after it began, excusing himself for not having come sooner on account of general illness, which his physician had called congestion of the brain, threatening apoplexy, and treated accordingly. I examined his ears; they appeared as

before, the naso-pharyngeal catarrh less, the Eustachian tubes pervious, the air heard on auscultation, the tick of a watch applied to the auricle or to different places on the head not perceived; the voice only when loud and at the ears. The sound of the tuning-fork from the glabella was heard well in either ear. Sounded near the ear the vibrations of a tuning-fork of middle pitch were distinctly perceived in each ear; but neither those of a very low nor those of a high tuning-fork were heard. All the notes sounded on the piano were heard with each ear.

The examination with the *galvanic current*, strictly according to Brenner's method, gave the following result:

Left ear, xx elements, 1000 units of resistance:

Ca C, whistling.

Ca D >

Ca O ——

An C ——

An D ——

An O whistling.

Right ear, up to xxix elements and 2000 resistances, only pain, no specific reaction.

In another sitting, two days later, the left ear gave the same reaction as above noted, and the right also gave the same formula with xxv elements and 1800 resistances.

This case shows that an acute aural catarrh, resulting from sea-bathing, passed over into a chronic state, which was improved by treatment, but was followed a year afterward, that is, three years and a half after it first set in, by a sudden attack of Ménière's disease, presenting all its symptoms except actual vomiting.

Affections of the inner ear consequent on *diseases of the brain* and its enveloping membranes, are certainly frequent. There are many canals leading from the cranial cavity into the labyrinth liable to be the channels for

the propagation of morbid action and its products, such as serous, hemorrhagic, and purulent exudations. That deafness is sometimes the result of the various forms of meningitis has long been generally known. The centre of audition or the acoustic nerves may be destroyed by meningitis and cerebritis without the occurrence of any changes in the inner ear. But if we are allowed to draw conclusions from the analogy of the ear with the eye, there will be, in the majority of cases, an extension of the morbid process in the organ of sense. Acute hydrocephalus produces deafness in some cases, blindness in others, and neither in most cases. The same may be said of meningitis cerebro-spinalis in its epidemic and sporadic form. These diseases run their course sometimes in a few weeks, and among the sequelæ deafness may be the only one to attract particular notice. Are we justified by that in considering the whole, or at least the primary and principal disease in these cases to be "*Otitis intima sive labyrinthica*"? We would certainly often make a false diagnosis. Does, on the other hand, no idiopathic inflammation of the labyrinth occur? Its existence, I think, should be as little denied as the existence of idiopathic retinitis which the ophthalmoscope now has transferred from the region of supposition into that of reality. I am ready to acknowledge the great merits of Dr. Voltolini for the description of "Otitis intima sive labyrinthica" (19, 20, 21), but must at the same time express my opinion that his views, although confessedly more restricted at present than they have been, are still too

extreme. I will first relate a case in order to base the discussion upon clinical data.

Case V.—February 22d, 1871, a healthy-looking girl (Carrie Stimus, of 128 Charlton Street, New York City), æt. 4½ years, was brought to the New York Ophthalmic and Aural Institute, with the following history:—The child had always enjoyed good health until last March, after which, during the course of three months, she sometimes ran anxiously to her mother crying that she was going to fall. June 13th she became ill, having headache, paleness of the face, nausea, vomiting, loss of appetite, general heat. She felt better toward evening, slept well until 5 o'clock the next morning, when suddenly she had intense headache, vomiting, convulsions in the nape of the neck (opisthotonus), convergent squint, and contractions of the muscles of the left side of her face. Although her head felt confused, she answered correctly when spoken to. Soon afterward she became comatose, but woke from time to time with violent convulsions. The symptoms reached their acme on the fourth day, lasted unabated until the sixth day, and from the seventh day they (headache, vomiting, convulsions of the neck, face, and eyes) disappeared gradually, and she was able to get out of bed by the eleventh day. She was, however, so weak that she could not walk for the next five weeks. When she could walk, she was so dizzy that she frequently fell down, after having made a few steps only, sometimes forward and sometimes sideways. This dizziness and uncertainty of gait diminished only very slowly, and even now, seven months after the attack, she is not entirely free from them.

On the second day of the acute attack it was noticed that the child had become completely deaf, and she has remained so ever since. No other symptoms on the part of the organs of hearing have been observed, nor was I able to detect any on examination when the child was brought to me. The tympanic membranes appeared normal, were movable, and the rushing of air into the tympanum on inflation could be heard by auscultation. No perception of any sound could be demonstrated. The examination with the galvanic current, which was made several times, gave no result.

The symptoms and course of this case answer to the description of meningitis cerebro-spinalis. As this disease mostly appears epidemically, sporadic cases of it may have been frequently described as meningitis simplex. There was, as far as I know, no epidemic of meningitis cerebro-spinalis in New York last year.

The first question which we have to answer with regard to this case is: Was, in the case just related—which is only an example of many similar ones on record—the ear-disease primary or consecutive? VOLTOLINI (19, 20, 21), and in conjunction with him G. REICHEL (22), strongly advocate the former opinion, and even go so far as to deny (Voltolini) or doubt (Reichel) the cerebral origin in the 13 cases of deafness in consequence of meningitis cerebro-spinalis epidemica reported by PROF. S. Moos (23). These cases of Moos were all observed in the wide-spread and well-marked epidemic which occurred in the valley of the upper Rhine in 1864. They were all diagnosticated and entered on the list as such by physicians who knew this disease well from their own extended experience. NIEMEYER's excellent monograph (24) describes the same epidemic, and some of the cases of Moos are also cases embodied in Niemeyer's treatise. I myself saw several of Moos's cases, and some others besides, occurring in the same epidemic, in which I took a great interest, principally on account of the fatal eye-disease which accompanied about five per cent. of the cases. Most of them which had been complicated with eye-disease I did not see till after their recovery from the

meningitis; but I also observed some during the course of the inflammation, while on visits to the principal towns in which the epidemic occurred. Exceptionally the eyes as well as the ears were affected, and blindness, together with deafness, remained after the recovery from the meningitis. The cases in which the eyes were affected showed exactly the same chain of general symptoms as those which were followed by deafness, and those in which neither the eyes nor the ears were implicated. Is there any reason for thinking those cases which were followed by deafness, instances of primary "otitis intima s. labyrinthica mistaken for meningitis"? Certainly just as little as for thinking the cases followed by blindness examples of primary ophthalmia interna mistaken for meningitis. The anatomical nature of the eye affection in epidemic cerebro-spinal meningitis is, as far as facts demonstrate, identical with that of the ear affection. In the vast majority of cases the eye loses its sight by purulent irido-choroiditis. Three *post-mortem* examinations of HELLER and LUCAE showed that in *cerebro-spinal meningitis purulent inflammation existed within the labyrinth.* In some cases the eye affection is simple exudative or plastic iritis, or irido-cyclitis, and their sight may be preserved or regained by an operation. In analogy to this, several cases are on record in which hearing was preserved or regained after the recovery from the meningitis. The inflammation in these cases must have been of a less destructive character, more plastic than purulent. Voltolini's objections, though

couched in most emphatic language, do not in the least invalidate Moos's views. I answer only two of them. Voltolini says that meningitis cerebro-spinalis with great preference occurs in adults, and has almost always a lethal termination. This statement is not founded on statistics. The cases of recovery from cerebro-spinal meningitis, in which blindness remained, were mostly children. This is the result of my own experience and of all the writers on the subject. The cases of recovery from meningitis in which deafness remained were, according to Moos, also mostly children. My conviction is, that meningitis is generally less dangerous in children than in adults. This conviction is based on the fact that neuro-retinitis, which is an extension of meningitis, and can be followed in its whole course, affords a better prognosis, quoad visum and quoad vitam, in the young than in the old. I could cite many instances to substantiate this, and it was also, if my memory serves me, the opinion of Von Graefe. This may help us to explain why we observe deafness and blindness acquired under meningitic symptoms more frequently in children than in adults.

As another argument in favor of the idiopathic inflammation of the labyrinth in the cases under consideration, Voltolini and Reichel point out that other nerves are not permanently affected, especially that facial paralysis has not been noticed together with deafness. This shows that the adjacent centres and trunks of both these nerves are not destroyed, and renders it evident that the cause

of the deafness must be in the destruction of the expansion of the auditory nerve in the labyrinth. But it does not prove that the ear-affection is primary. In what percentage it occurs in cerebro-spinal meningitis we know not, as it must have been frequently overlooked in the lethal cases. It is mentioned, however, by all writers on the disease under consideration, and WUNDERLICH (Arch. f. Heilkunde VI., 268 etc., Med. Centr.-Blatt, Bd. III., p. 349) says: "Headache, stiffness of the neck and back are always present; in addition, deafness in one or both ears is a decisive though not constant symptom." He is of opinion that the occurrence and propagation of cerebro-spinal meningitis argue in favor of its infective nature. The affection of the membranous labyrinth, purulent inflammation, then might be of a pyæmic nature. The eye affection is certainly of that kind, as is pointed out by Dr. Joseph Jacobi (Arch. f. Ophth., XII., 3, p. 156–167). Not long before his paper I had given a short description of the symptoms of the eye-affection in meningitis cerebro-spinalis (Med. Centr.-Blatt, 1865, p. 513, etc.), and termed it hyperplastic or sarcomatous choroiditis, a very inappropriate name, which I had chosen, to remain in conformity with the leading text-book on Ophthalmology at that time, namely, Stellwag's, where this disease, though mixed with heterogeneous conditions, had been described under that name; but I soon convinced myself that it was identical with the purulent irido-choroiditis which occurs in puerperal fever, and is evidently of a pyæmic or metastatic

nature. I had an opportunity to observe during life three eyes thus affected, and to examine them microscopically after the death of the patients. (Described, Arch. of Ophth., Vol. XIII., 1, p. 127–181.)

Sight in puerperal fever is sometimes destroyed in one or both eyes in a very short time, for instance in twenty-four hours, by purulent choroiditis. I should not be surprised if hearing were destroyed in a similar way; but I am not sufficiently acquainted with the literature to support this supposition by reported cases. Whether other infectious diseases act in the same way, I know not. Pathology has shown that in cerebro-spinal meningitis the hearing may be impaired or destroyed by other processes than purulent otitis labyrinthica, namely, suppuration in the fourth ventricle, or along the course of the auditory nerve, or in the tympanic cavity. In the same way the eyes may suffer from other diseases than purulent choroiditis. Neuro-retinitis has been observed (Schirmer), and this indicates an immediate extension of the inflammatory products from the arachnoid cavity along the optic nerves into the eye-ball. A similar propagation of the inflammatory products into the inner ear seems to be very probable, but has not yet been demonstrated, as far as I know. It would not produce total deafness, at least not in a short time, if we may judge of it from neuro-retinitis, where sometimes after months only the eye becomes blind by progressive atrophy of the optic nerve. The principal anatomical lesion in the eye produced by cerebro-spinal meningitis is purulent choroiditis, disconnected with the

purulent inflammation within the cranial cavity; so the principal lesion in the ear is probably—I say probably because I know only of three corroborating post-mortems—a purulent inflammation in the labyrinth, likewise disconnected with the cranial cavity. This would explain why facial paralysis so rarely accompanies it.

A remarkable and somewhat surprising feature of this ear affection *is the frequency of its occurrence in both ears.* Instead of attempting to explain this by an unknown predisposition of the ears to simultaneous or sympathetic inflammation, I prefer to state that frequently both eyes also are affected and become totally blind in cerebro-spinal meningitis, as for instance was the case in nine out of the eleven of J. Jacobi's patients.

If all these facts derived from the epidemic cerebro-spinal meningitis be true, they will perhaps, with certain modifications or restrictions, be valid also for the *sporadic* occurrence of the same disease. That this holds good for purulent internal ophthalmia in sporadic cases of puerperal fever I know by personal observation.

This leads to the question whether such cases as the fifth above described—where, under the full array of the symptoms of acute meningitis, or cerebro-spinal meningitis, deafness ensues—are primarily meningitides, or otitides. The possibility of the occurrence of purulent otitis interna as a primary disease cannot, *à priori*, be denied, though it has not yet been confirmed by autopsy, except in traumatic cases. If it occur, it in all probability causes severe cerebral symptoms, as do many cases of

otitis purulenta media. To my mind, it must be exceedingly rare. But since I am quite as unable to disprove by anatomical investigation the frequency of its occurrence as its advocates are to prove it, they may allow me again to arrive at probabilities by analogy. Purulent choroiditis is hardly known as an idiopathic disease; it is either of traumatic or pyæmic origin, or communicated from the suppurative inflammations of adjacent tissues, principally from the cornea and iris, rarely from the orbit. The labyrinth is a structure analogous to the parts of the eye lying behind the crystalline lens and iris, but better protected from external injurious causes. The mere, though undeniable possibility of the occurrence of a certain disease, is not sufficient to prove its frequency. Nobody, for instance, could doubt, *à priori*, the occurrence of a blennorrhœa of the male urethra not acquired by infection; but what confidence do we place in the assertions of those "respectable" gentlemen who swear they could not have caught it from women? In the same way, unless convinced by facts to the contrary, I am inclined to think otitis labyrinthica purulenta a secondary disease, either of a traumatic or pyæmic origin, or imparted from the neighboring tissues, the bones, the membranes of the middle ear, or the contents of the cranial cavity.

The principal argument of Voltolini in favor of idiopathic otitis labyrinthica is the recovery of the general health of the patients. Meningitis of any kind, he says, is a lethal disease in the immense majority of the cases, if not always. The opinions of physicians on the question of

the mortality in meningitis are divided. Some pretend to have observed many recoveries, but their opponents allege that then the diagnosis was incorrect. Blindness and deafness are mentioned as frequent sequelæ of meningitis. In regard to the former I can state by manifold observation that I have seen all possible degrees of impairment of sight supervene after sporadic meningitis. During the acute attack, we see with the ophthalmoscope neuro-retinitis, which, after the recovery of the patient, gradually passes over into a more or less advanced state of atrophy of the optic nerve. To cite one example, I may mention that a boy five years old, the son of a physician of this city, suffered from acute meningitis two years ago, with neuro-retinitis and paralysis of the abducentes. He recovered from the severe cerebral symptoms in the course of thirteen days, while the neuro-retinitis slowly disappeared, leaving the ophthalmoscopic appearances of atrophy of the optic nerves, with very good vision in one eye and moderately good vision in the other. Convergent squint, which had developed from the paralysis of the external recti, passed away almost completely in the course of these two years. Meningitis is more frequent, and its prognosis quoad vitam vastly more favorable in children than in adults. It is, therefore, not surprising that blindness or deafness acquired from meningitis is more frequent in children than in adults.

With regard to the diagnosis whether the otitis labyrinthica or the meningitis is the primary affection, I raise only the following question: If the same complex of cerebral

symptoms in some cases produces deafness, in others blindness, and in many others neither, why should we call the first group otitis labyrinthica mistaken for meningitis, whereas in the second group the dependence of the ocular affection on the cerebral disease can be demonstrated?

Thus far we have seen that there are in the labyrinth inflammatory processes of very different origin and nature; primary and secondary, hemorrhagic, traumatic, exudative—with the symptoms of increased pressure—and purulent. Not wishing to interrupt too frequently the context of this paper by the history of cases, I described only such observations as appeared to be representative of the varieties of otitis interna. There are, however, many cases coming before the aural practitioner where it is not easy, if possible at all, to make an exact diagnosis. Both practical and theoretical interests require that such cases should not be passed over in silence, but, on the contrary, conspicuously pointed out as problems awaiting their solution from further labor. Prompted by these motives, I shall now present a condensed history of all the cases of actual, probable, or possible inflammatory affections of the labyrinth that came under my notice during the twenty-seven months of my sojourn in New York. I omit the observations of hyperacusis, diplacusis, and defects in the range or amplitude of audition, confining myself to the report of those cases in which the hearing had been more or less impaired. Their number was 42 out of 1673 patients suffering with ear diseases, that is 2½ per cent.

Cases of injury may cause gradual or sudden, partial or complete deafness.

CASE VI.— ——, æt. 24 years. Deaf in right ear on which he was frequently *struck* when young. No other cause assignable. Left ear healthy. On physical examination both outer and middle ears alike and healthy.

The lesions resulting from boxing the ears may be either in the inner or the middle ear. As in the foregoing case, no abnormality was detected in the sound-conducting apparatus on physical examination, it seems natural to assume that the labyrinth had suffered from hemorrhage and inflammation with or without fracture of its bony parts. The occurrence of traumatic hemorrhage in the labyrinth without fracture, appears plausible when we remember the hemorrhages in the cranial cavity, and the isolated ruptures of the inner membranes of the eye, especially of the choroid, produced by blows on the head or eye, and unconnected with any solution of continuity in the skull or the fibrous capsule of the eye (see Archives of Ophthalmology and Otology, I., 1, p. 149, etc.). This explanation is borne out by the post-mortem examination of a case of gun-shot injury to the head, described by S. Moos in a subsequent paper of the present number of these Archives.

CASE VII.— ——, 10 years old, lost hearing two years ago from a *fall*. Vomiting, hemorrhage from left ear, perhaps also from right. He has retained slight perception of the loudest sounds. On physical examination no abnormality.

CASE VII.— ——, 11 years old. When seven years old, *fell down stairs*, had convulsions, followed by delirium for a month. Deafness

was discovered a week after the injury. It is complete. No changes in outer and middle ears.

Case IX.— ——, æt. 21. When 2 years old, *fell down stairs*, and became deaf from it. No perception of sound. No abnormality on physical examination.

These three latter cases of severe injury by a fall on the head are analogous to the second, and the cases of Politzer and Voltolini, quoted above. As they were followed by severe cerebral symptoms and permanent deafness, hemorrhage and subsequent purulent otitis interna in consequence of fracture of both petrous pyramids, is to be assumed on the ground of the post-mortem conditions found in the cases of Politzer and Voltolini.

Case X.—*Ménière's disease after childbirth.* Mrs. ——, æt. 32, has been married for ten years. In her first confinement, nine years ago, she suffered from vertigo, vomiting, and haziness before the eyes, for about two months. The hearing was unimpaired. Her second confinement, seven years ago, passed without any abnormal symptoms. In the third, four years ago, she suffered again, but only for some days, from vertigo and vomiting, being free from any disturbance of hearing. Six weeks after the birth of her fourth child she ate, one evening, a large piece of ham. In the night she had vertigo, nausea, and vomiting. On trying to get out of bed in the morning, she felt exceedingly dizzy, and staggered from one side to the other. She vomited for twenty-four hours, but never lost her consciousness. Her hearing was very dull. Heaviness of the head, vertigo, and the uncertainty of gait, have continued up to this moment. She complains of her head not being fast on her shoulders. When she is not supported in the dark, she falls. In walking she reels like a drunken person, and must frequently be guided. She has only quantitative perception of sound, and is unable, even with the most powerful hearing-trumpets, to understand words. These instruments cause an unpleasant sensation in her head. The intonation

of her voice has much changed in these six months, though she still speaks distinctly. The tympanic membrane and the outer and middle ears show no signs of abnormality. On examination with the galvanic current she had with 10 elements and 1000 resistances a sensation of ringing at the cathode closure in the left ear, and the same sensation in the right ear with 25 elements and 2000 resistances at the cathode closure.

This case, as well as the third, fourth, and forty-second, is a fair representative of Ménière's disease, the symptoms of which have been described at length in the first part of this paper. Childbirth produces general anemia, but may, at the same time, occasion *local* hyperemia, stasis, extravasation, and inflammation. The symptoms of our case lead to the assumption that the latter took place in both labyrinths. It is worthy of notice that the patient had the same phenomena, though in a less degree, during two previous confinements.

The following group of cases are analogous to the fifth reported in the context, namely, loss of hearing during the course of some cerebral affection, which in most cases corresponds to the description of simple meningitis. I shall limit myself to short abstracts of the histories.

Case XI.— *Cerebro-spinal meningitis.*— ——, æt. 17. When 7 years old had meningitis, in which opisthotonus was a prominent symptom. During the disease he became deaf on both sides, and remained so ever since. No other sequel of the disease was left behind. On examination I found the deafness complete, and no change in the outer and middle ears.

Case XII.— ——, æt. 5. *Meningitis* when 1½ years old, which rendered him completely deaf. No abnormality on physical examination.

Case XIII.— ——, æt. 8. *Meningitis* when 5 years old, which

left total deafness. No physical abnormality in outer and middle ears.

Case XIV.— ——, æt. 2½. *Meningitis*, with consequent total deafness when eight months old. No physical abnormality in outer and middle ears.

Case XV.— ——, æt. 3 years. *Meningitis* when 13 months old. Deaf ever since. Seems to hear very loud noises. No abnormality on physical examination.

Case XVI.— ——, æt. 7. *Meningitis* when 1½ years old. Deaf ever since. Tympanic membranes somewhat sunken.

Case XVII.— ——, æt. 5. *Meningitis* when 3 years old. After recovery hardness of hearing, which constantly increased. Is now completely deaf. No change in outer and middle ears on physical examination.

Case XVIII.— ——, æt. 5. When 4 years old became severely *sick* (*meningitis*), and was confined to bed for 7 weeks. Hearing was failing during the illness, and totally lost after the convalescence. No abnormality on physical examination.

Case XIX.— ——, æt. 4½. When 3½ years old had sometimes, in the course of 3 months, attacks of sickness and vomiting, then severe "gastric fever," with headache, vomiting, and somnolence. It lasted four weeks, and left complete deafness. Outer and middle ears normal.

Case XX.— ——, æt. 2½. When 1½ years old had meningitis, which rendered him deaf. No perception of sound. No physical abnormality.

Case XXI.— ——, æt. 8. When 6 years old had intense fever (*meningitis*). His head ached and was very red. No vomiting, delirium, or eruption. He was constipated. Ice was applied to his head. On the tenth day he woke up in the night, when all around was quiet, and asked repeatedly "that the music might be stopped." Soon after he fell asleep again, and on awaking in the morning he was found to be completely deaf. He recovered from his fever in a week, but had an uncertain, staggering gait long afterward. He was persistently treated in different ways; I mention, in particular, the employment of electricity for six months. When he presented himself to me, he gave no evi-

dence of any perception of sound. There was normal wax in both ears; the tympanic membranes were whitish at their lower borders, the handles projecting, and the tonsils enlarged. Inflation normal. The boy had gradually lost his speech.

Case XXII.— ——, æt. 10. When 9 months old had *meningitis*, with convulsions, unconsciousness, vomiting. Completely deaf afterward. No change in outer and middle ears found on examination.

These thirteen cases, it seems to me, would all be claimed by Voltolini as belonging to his otitis interna. I have above given the reasons why I must differ from his views, and take such cases as these to be primary meningitis with consequent deafness resulting from purulent inflammation in the labyrinth. I agree with Voltolini's assertion that they are not rare, for he has in his own aural practice observed over 70 cases, and the number of 13 cases which, in the course of two years and a quarter, came under my own notice, corroborate his statement. His cases, like mine, were all sporadic affections, no epidemic of cerebro-spinal meningitis having occurred either in Silesia or New York. The course of the acute disease—the severe cerebral symptoms—was usually between two and three or four weeks, exceptionally (Case XVIII.) some weeks longer. This coincides, as far as I can ascertain, with the average duration of meningitis simplex.

In the great majority of the cases the hearing was completely destroyed during the febrile period; in others it was incomplete during the course of the acute meningitis, but became complete afterward (Case XVII.), or seems to have remained incomplete (Case XV.).

That the cochlea is the principal seat of the aural lesion follows from the total or almost total destruction of hearing. One case (the 21st) was remakable on account of the subjective sensation of musical sounds which preceded the loss of hearing, a proof that excitation of the acoustic nerve was previous to its paralysis.

The destruction of hearing was rapid in many cases, just as the rapid destruction of sight, sometimes in less than twenty-four hours, is one of the prominent symptoms of purulent choroiditis.

The external and middle ears in all but one of the cases that came under my notice, were found normal. The one exceptional case (the 21st), showed symptoms of chronic otitis media, resulting from pharyngo-nasal catarrh with enlarged tonsils.

Closely allied to the deafness in consequence of acute meningitis simplex or cerebro-spinalis, is that in connection with *eclampsia infantum.*

Case XXIII.— ——, æt. 4. When nine months old had *convulsions* and other cerebral symptoms. Since that time hard of hearing and subject to *epileptic fits.* Was worse than at present. Hears a little with the left ear, nothing with the right. Articulates a few words. Result of physical examination negative.

Cases XXIV. and XXV.— ——, æt. 6, and ——, 4 years. Two brothers; had *convulsions* in their first year. Both heard well before, but became deaf during the attacks, and remained so ever since. Nothing abnormal on physical examination.

Case XXVI.— ——, æt. 5. Had *convulsions,* and lost his hearing when three years old. Deaf-mute. Low mental development. Membranæ tympanorum dull, otherwise normal.

Case XXVII.— ——, æt. 3½. *Convulsions* when six months old. No sign of hearing afterward. Deaf-mute. Result of physical examination negative.

Case XXVIII.— ——, æt. 2¾. Was in good health until nine months ago. He went to bed without any symptoms of disease, and at midnight awoke with *convulsions* in his arms and legs, but no opisthotonus, no spasms in the eyes, no nausea, no vomiting. He was crying, and his skin was very hot. This attack lasted for about a quarter of an hour, when the child fell asleep again. He slept well until morning, when he awoke apparently quite well, but the mother noticed that he could not hear. A month later she brought him to me. I found no abnormality in his ears so far as physical examination was applicable, but all perception of sound was lost. He was treated for some time with the galvanic current according to Brenner, but never showed the least evidence of hearing.

This last case is analogous, in regard to the suddenness of the destruction of hearing, to the first in this paper. There was hyperæmia of the brain, as the crying and the heat of the skin would indicate. The sudden total and permanent loss of hearing must be ascribed, as seems to me, to *apoplexy* either in the common nervous centre of audition or *in both labyrinths*. The latter appears more probable, as the former could hardly have been limited to the auditory nerves, and would have produced cerebral symptoms of longer duration. The suddenness of the attack and the immediate deafness, which is nearly always complete and binauricular as it seems, are very peculiar features of these cases. The causes of eclampsia infantum are manifold. They frequently consist in irritation of a peripheric nerve transmitted to the medulla oblongata, but when they are followed by paralysis or

anæsthesia of some part of the body, or impairment or destruction of one or several senses, they have been considered as a symptom of an affection of the brain (*Chomel: Pathologie Générale*, 3d ed., p. 396). Whether they are connected with hyperæmia or anæmia of the brain is still an open question (Niemeyer's Lehrbuch). The sudden and mostly complete destruction of hearing, without any other lasting consequences, leads forcibly to the conclusion that an acute lesion, hemorrhagic or serous apoplexy, takes place in the labyrinth, and destroys the function of the auditory nerve by pressure and the consequences of the exudation. Hyperæmia, stasis, extravasation, and serous effusion being symptoms of inflammation, producing changes of nutrition, I think we have to classify these cases among the inflammatory affections of the inner ear. Anæmia may also produce impairment or loss of sight and hearing. I am not prepared to enter satisfactorily into the details of this subject, the knowledge of which cannot fail to be extremely important in regard to the pathology and therapeutics of nervous diseases in general.

The close connection between eclampsia and epilepsy is exemplified in Case 23, where the child, after the convulsive attack, remained subject to epileptic fits. The destruction of hearing in this case was also incomplete, and the presence of other cerebral symptoms is mentioned, which makes the case a connecting link between those resulting from pure convulsions and those from meningitis.

A question of importance is, whether the *labyrinth affection in these cases is primary or secondary.* The one or the other may be admitted. Supposing that *primary hyperæmia* and serous or hemorrhagic effusions in the *inner ear* take place, then the acoustic nerves will be irritated, and excite the motor nerves so much the more readily as their fibres are directly connected with the gray matter of the medulla. Pushing consequences of this supposition further, we may say that the convulsions must cease as soon as the effusion in the closed cavity of the labyrinth has become so abundant as no longer to excite but to paralyze, by pressure, the auditory nerves. On the other hand, it is just as conceivable that *general* hyperæmia, or *cerebral* hyperæmia, produces small ecchymoses in the labyrinth only, thereby impairing the sense of hearing without causing lasting cerebral symptoms. Thanks to the ophthalmoscope, we are familiar with such a condition in the retina.

Most writers on otology say nothing or little on the deafness in connection with eclampsia infantum. The following statement is found in ITARD'S text-book (24): "La paralysie du nerf acoustique à la suite des convulsions est une cause de surdité fort rare chez l'adulte, et très-fréquente dans le premier âge. Lorsque l'ouïe se perd dans les trois ou quatre premières années de la vie, c'est presque toujours à la suite des convulsions. Un grand nombre de sourd-muets doivent leur infirmité à une pareille cause. Il est digne de remarquer que ce sont les convulsions les moins fortes, les moins prolongées, qui

produisent le plus communément cet effet. Nombre d'enfants devenus sourds vers l'époque de la dentition, et pour lesquels j'ai été consulté, avaient pour la plupart cessé tout-à-coup d'entendre, immédiatement après un léger mouvement convulsif."

Some observations have recently been published by Dr. Hughlings Jackson (25), Köppe and Schwartze (26), which seem to demonstrate that reflex epilepsy may be occasioned by inflammatory diseases of the middle and inner ear. The epilepsy disappeared after the successful treatment of the ear affections. In consequence of such facts it would seem natural enough to assume that a sudden primary irritation of the labyrinth might cause eclampsia in early childhood, the age so liable to convulsions from any cause. In spite of all this I am inclined to believe that cerebral congestion is the primary affection, which by extension into both the labyrinths and the production of serous or hemorrhagic effusion into them, irritates the acoustic nerves and causes convulsions by reflex action on the medulla. When the sero-sanguineous effusion is so abundant as to annihilate, by pressure, the conductibility of the auditory nerve-fibres, the reflex convulsions cease, and hearing may be destroyed forever. When the conductibility of the auditory nerve-fibres is not annihilated but only blunted, hearing is only impaired. As such attacks of congestion are liable to recur, they may also occasion repetitions of the convulsions. I would call the affection true reflex epilepsy with an aura beginning in the labyrinth. Case 23 may exemplify this.

Another group of cases are those of deafness occurring in the course of *severe fevers*, especially *typhus*, *typhoid*, the *acute exanthemata*, and *certain eruptions* on the head and neck, without discharge from the ear or any conspicuous symptom of otitis media.

Case XXVIII.— ——, æt. 10 years. When 8 years old had *typhoid fever*, on the third day of which deafness on both sides ensued, which has remained unchanged. Hears now the watch only on contact with the ear. Membranæ tympanorum bluish, sunken.

Case XXIX.— ——, æt. 19 years. When 11 years old had *typhoid fever with convulsions*. Since then has gradually lost his hearing. The deafness is complete on both sides. Membranæ tympanorum normal, tympanic cavities inflatable.

Case XXX.— ——, æt. 22 years. When 18 years old became deaf from *typhoid* fever. No perception of sound. Result of physical examination negative.

Case XXXI.— ——, æt. 15 years. Deaf from *typhoid*. No perception of sound. Physical examination negative.

Case XXXII.— ——, æt. 24 years. When 22 years old had *typhoid* fever, which lasted three weeks, and *rendered him deaf in the course of two hours*. He heard, afterward, humming noise in one ear and nothing at all in the other. When he came under my notice he had no perception of sound whatsoever. The tympanic membranes appear thickened, the relief of the right somewhat irregular; in the left there was a small white spot (chalky deposit).

Deafness, so common a symptom in typhoid and typhus fevers, may be caused by otitis media, hyperæmia, exudation, and hemorhage in the labyrinth, and, perhaps, also, by some change in the nutrition and function of the brain. Most of the above cases show symptoms of otitis media, but the complete, or almost complete, deafness in most cases, and the pretty sudden loss of hearing in the last

case, speak for the participation of the labyrinth in the morbid process. Hemorrhage in the labyrinth would seem the probable cause of it, and has, indeed, repeatedly been found by autopsy of persons deceased from typhoid and typhus fevers. My opinion is that in most cases otitis media, propagated from the affection of the naso-pharyngeal region, occasions secondary hyperæmia, hemorrhage, and inflammation in the labyrinth and partial or complete deafness, according to the degree of destruction of the expansion of the auditory nerve. I am aware that in some autopsies of persons who succumbed to typhoid fever while they were deaf, changes in the inner ear have been absent. In such cases nothing is left for explanation but the unsatisfactory supposition of cerebral or anæmic deafness. This subject might be cleared up, if aurists would work in conjunction with the attending physicians of hospitals where typhoid patients are numerous. They should examine, during the course of the disease, the parts of the hearing organ susceptible of physical exploration, state the amount of hearing, and, in case of death, investigate the middle and inner ears together with the auditory nerves and the brain. Typhoid and typhus seem to me to furnish the most available opportunities for making inquiries into the inflammatory changes in the inner ear.

Case XXXIII.— ——, æt. 3½. When 6 months old had *measles*, unaccompanied by discharge or any other symptoms in the ears, but has been deaf from that time. No perception of sound. Membranæ tympanorum and middle ears normal, as far as can be ascertained.

Case XXXIV.— ——, æt. 10. When 3 years old had scarlet fever, but no otorrhœa. In the period of convalescence her hearing began to fail, and was lost in the course of one week. No perception of sound. Nothing abnormal on physical examination.

The acute exanthemata, still more than typhoid fever, are liable to be complicated with catarrhal or purulent otitis media. In exceptional cases, however, we notice that more or less sudden and complete loss of hearing takes place during their course, and in such cases a sero-hemorrhagic inflammation of the labyrinth must be assumed as the probable cause of deafness.

Case XXXV.— ——, æt. 26 years. When 2 years old had *erysipelas*. Deaf and dumb from that time. The only sound he perceives is that of a tuning-fork held close to the auricle. Has tinnitus in the right ear only. Membranæ tympanorum normal.

Case XXXVI.— ——, æt. 28 years. When 20 years old was suddenly attacked with *pustular* and *eczematous* eruption, extending over the entire head, and accompanied by fever and delirium. Both ears were swollen. Hearing diminished on the fourth day of the eruption. The eruption disappeared on the eighth day very rapidly after the application of a red ointment. After that time hardness of hearing has always been as it is now. No conduction of sound through the cranial bones. Hears nothing with the left ear, but with the right he hears faintly the watch on contact with the auricle, the tuning-fork at the distance of half an inch, the musical box at 4 inches. No abnormality whatever on physical examination of both ears.

Case XXXVII.— ——, æt. 3 years. When 2 years old had eruption over the whole head, which healed completely in a week, but left the child deaf ever since. Six weeks later had meningitis, and remained feeble on his legs a long time afterward. Hears only loud noises and loud music. No abnormality on physical examination.

I am not aware that post-mortem examinations of the

labyrinth have been made in persons deaf from and dying of erysipelas or acute pustular and eczematous eruptions extending over the head and involving the ears. I must, therefore, resort to the hypothesis that the inflammation extended to the labyrinth, and caused, by the high internal pressure, serious impairment or annihilation of the functions of the auditory nerves.

The following observation is unique in my experience:

Case XXXVIII.— ——, æt. 7 years. When 1 year old, had meningitis, which was preceded for one week by repeated vomiting. During the fever she was unconscious and vomited frequently, but had no convulsions. After recovery she felt weak for some time, but heard well.

Four months later she had a large, white, hard intumescence below both ears and around the whole anterior part of the neck (*angina Ludwigii*), but not in front of the ears. It caused great distress in swallowing, which continued, in a less degree, some time after recovery. While the swelling existed the child was crying, nearly uninterruptedly, and often put her hands to her ears. At the beginning of the third week the swelling disappeared rapidly—in twenty-four hours, as the parents allege—after the application of an irritant ointment. After this the child never showed any signs of hearing. When she was brought to me she was deaf-mute, had no perception of sound whatever; physical examination revealed no abnormality in her hearing apparatus.

Toynbee mentions nervous deafness occurring in the course of mumps, but in our case the parotid gland was unaffected. I do not know whether the affection of the throat and ears in Case 38 was connected with diphtheria; I prefer to give its history without comment.

Case XXXIX.— ——, æt. 28 years. Became hard of hearing 10

years ago after the *birth of her first child*, and got worse with every subsequent childbirth;—had four children. Does not hear the watch at all, understands loud voice at two feet in the one and at three feet in the other ear. On physical examination of the outer and middle ears nothing abnormal.

Case XL.— ——, æt. 40 years. Became hard of hearing in her first *childbed*, when 18 years of age, without pain or inflammatory symptoms. She now hears best when there is a great deal of noise around. Tympanic membranes normal. Drums inflatable. No watch heard, loud voice at 1′ and 2′ respectively.

Cases of *hardness of hearing after childbirth* are not rare. They are to be distinguished from those brought about by puerperal fever, which are, in all probability, of a pyæmic nature. The hardness of hearing in the former, of which the two related cases are examples, may be accounted for by anæmia. They have an analogon in the amblyopia after childbirth. It is conceivable, however, that also serous effusions or ecchymoses in the labyrinth may play a part in the production of this kind of deafness, and, therefore, I did not omit to mention here the two observations which came under my notice. This latter supposition is strengthened by our 10th case, in which we saw a well-marked instance of Ménière's disease occur after childbirth.

Case XLI.— ——, a healthy man, æt. 51, had a *sunstroke* when 49 years old. During the succeeding three weeks all *noises* around him appeared to be immensely *intensified* and very *disagreeable*. Then he noticed that his left ear had become entirely deaf, his right somewhat hard of hearing. This state has remained unchanged up to the present day. On examination I found that his outer and inner ears were normal. With the right ear he could hear ordinary conversation easily

enough, and the ticking of the watch at the distance of 1′ (instead of 10′). With the left he hears nothing at all, neither the watch when applied to the auricle or on the skull, nor the tuning-fork from any part of the head, while the tuning-fork is well heard from the glabella with the right ear, and its sound intensified when this ear is closed.

The hyperacusis observed in this case for three weeks after the action of the heat-stroke shows that irritation of the acoustic nerves was present. Serous or hemorrhagic inflammation, producing increase of intra-auricular tension, was most likely the initial stage of the affection in the labyrinth. In the second and final stage the conductibility of the auditory nerve-fibres seems to have been destroyed, by pressure, in one ear completely, in the other only partially. As hyperæmia of the meninges has been found in persons who died from heat-stroke, this explanation is somewhat borne out by pathological data. In glaucoma the increase of intra-ocular tension, by serous or hemorrhagic inflammation, produces also symptoms of irritation of the optic nerve in the first stage, and afterward diminution and destruction of sight.

The following case is an instructive illustration of the manner in which *syphilis* may affect the inner ear.

Case XLII.—Mrs. S. D——, æt. 42 years, suffered, in May, 1870, for some weeks from attacks of headache, nausea, and vertigo; all the objects around her seemed to vacillate. Her physician ordered her ice applications to the head. In July she had pharyngitis, in August a red papulose eruption over her whole body. On December 23d, 1870, she suddenly had severe headache, dizziness, and nausea, but no vomiting; she had intense tinnitus and rapid diminution of hearing. She

could not remain up. When she lay on the side, her bed and room seemed to tilt from one side to the other, like the rolling of a ship; when she lay on her back, her room balanced up and down, like the pitching of a ship. On every trial to get up she became so dizzy that she fell down, sometimes forward, sometimes sideways, with no tendency to fall in a determined direction. In the course of a week her hearing had become so dull that she could not understand the loudest speech.

After these symptoms had continued for three weeks, she suddenly felt, on Jan. 12th, 1871, an increase of headache and vertigo, and all the objects around her appeared to be shrouded in a thick fog. At the same time she saw bright sparks shooting before her, a wavering of the surrounding objects (Flimmern), and all possible colors appearing and vanishing in succession. These photopsies and chromopsies lasted only for a few minutes at a time, but often recurred during the course of three days, after the lapse of which they never returned. She felt severe pain in her eyes, forehead, and temples, and her sight grew dimmer from day to day. She was admitted to the N. Y. Ophthalmic and Aural Institute on Jan. 18th. The symptoms of syphilitic sore throat and papulose eruption over the whole body were very marked. She was emaciated, had a dirty lead-colored complexion, complained of violent headache, vertigo, and sickness, had no appetite, and slept badly. Both eyes were red and painful, the posterior surface of both corneæ dotted, the anterior chambers muddy, the irides discolored and swollen, the pupils irregular by many posterior synechiæ. The fundus oculi invisible, the tension of both eyeballs unmistakably increased (T_1). She complained more of distressing noises in her head than of anything else. She could barely count fingers, and was unable to understand one word even when shouted into her ears. She had, however, perception of sound and a complete visual field on both sides. The apparent motions of surrounding objects continued, but were least pronounced when she lay on her back. She therefore remained very quiet in this position.

She was ordered an aperient after that, gr. $\frac{1}{2}$ of calomel with gr. $\frac{1}{6}$ of opium, to take three times a day, and ℥ i of mercurial ointment to be rubbed into the skin of her limbs, also three times a-day. Five leeches

were applied to each temple in the evening, and the after-bleeding was kept up for two hours. Under this treatment her eyes improved very rapidly. The tension of the globe became normal already the next day. The synechiæ all torn, the episcleral injection, and the exudations in the anterior chambers gradually disappeared, and when the fundus had become visible, the existence of neuro-retinitis could be diagnosticated despite the diffuse haziness in the vitreous body. No blood-stain was visible anywhere, nor could exudative or atrophic patches be detected in the choroid or retina. Her sight improved in the same proportion. Her hearing made only very slow progress. After four weeks' treatment the general symptoms—the headache, the skin and throat affections—had almost disappeared. She could be up without falling, but staggered in walking, felt more or less dizzy at times, and complained still of the incessant though less distressing noises. She was able to understand loud voice. The ticking of the watch was not heard. She remained under treatment as an out-patient, taking iodide of potassium half a drachm daily, and was electrized.

The reaction of the galvanic current was the following:

Right ear:—No reaction, until with XXV elements, and 1000 units of resistance.

Ca C—ringing.

Ca D>30 seconds.

Ca O—

An C—

An D—

An O—ringing>20 seconds.

Left ear:—

XXV elements, 1000 units of resistance.

Ca C—ringing, loud and of long duration.

Ca D>

Ca O—

An C—ringing faint and short.

An D>

An O<

She was electrized from Jan. 24th to March 29th, without any change in this formula.

On April 22d I made the last examination, and noted the following conditions: Both corneæ and irides normal, S=$\frac{20}{40}$, and Tn in either eye. Both pupils round and movable. Both optic discs still hyperæmic, their margins partly indistinct, and the adjacent portions of the retinæ somewhat swollen. She hears no watch, R loud voice at $\frac{1}{2}'$, L. 1'. No tuning-fork is heard from the skull. She hears a tuning-fork of medium pitch vibrating before each ear; tuning-forks of high and low pitch are not heard. She does not hear the four highest (e''''–a'''') nor the four lowest (A^3–D^1) tones of a piano, hears the octave next to the highest tones, and following the lowest tones only feebly, the middle octaves best of all, though less far than a good ear. On Valsalva's experiment the sound of a short beat, free from any râle, is heard with the auscultation tube on both sides. A small quantity of normal wax in both ears. Tympanic membranes perfectly normal. The skin clear, complexion healthy; she has gained in weight, can walk about alone, but is still subject to dizziness. The noises continue, but are less distressing. Her appetite and sleep are good.

Of all the preceding cases this last one is, theoretically, and in consequence also practically, the most important. A person under the influence of constitutional syphilis shows the manifestations of this infectious disease successively in different organs; besides the throat and skin affections, the head is attacked in a way as we have seen it in connection with internal ear disease—pain, nausea, dizziness. After that there appeared a severe sudden attack of the same brain symptoms, in connection with well-marked disturbances of equilibrium, excruciating noises in the ears, and almost total deafness; a sudden

outbreak of Ménière's disease as complete as it can be seen. Three weeks later another attack of the cerebral symptoms ushered in severe internal eye disease, without interfering with the persistence of the aural difficulty. As it is impossible to observe directly the structural changes in the inner ear, it is of the greatest importance that we now are able, thanks to Helmholtz's immortal invention, to witness the changes within the visual organs which destroy sight under the general symptoms, in the same constitutional disease under which hearing is destroyed in the same patient. These changes consist in acute inflammation of the choroid and iris, with abundant sero-albuminous exudation into the humors of the eye. Is it not the most natural inference to presuppose the same conditions within the inner ear, viz.: acute inflammation with sero-albuminous exudation into the liquid of Cotugno? The increase of internal pressure demonstrable in the eyes, and presupposed in the labyrinths, would explain the symptoms of irritation of the acoustic nerve—tinnitus and disturbance of equilibrium. The parenchymatous inflammation overloading the vibratile parts of the inner ear, the membranes of both fenestræ included, with blood and serous fluid, must impede the ready transmission of the sound-waves to the auditory nerve in the same way as the same inflammation impedes the course of light-waves on their way through the eye. The brain, most likely, is in the same condition of hyperæmia and œdema, as may be inferred, not only by the cerebral symptoms, but also by the presence of hyperæmia and œdem-

atous swelling of the intra-ocular ends of the optic nerves and their vicinity, this œdema being nothing but a direct extension of the same fluid superabundantly collected in the arachnoid cavity of the brain. This hyperæmia and œdema are usually seen to persist longer than the gross changes in the tissues—pharyngitis, syphilides, iritis, etc. Their longer duration in the brain and labyrinth, although not directly seen as in the eye, may be inferred by the long persistence of the cerebral and aural symptoms. Any degree of sight, and probably also any fraction of the normal hearing power, may be preserved or regained in syphilitic inflammation of the eye and inner ear, according to the more or less complete absorption of the parenchymatous and free exudations, and the degree of atrophy of the nerves of special sense, which is apt to follow upon this disease.

In regard to the ear affection in syphilis I should not omit to quote an instructive passage from Jos. Gruber, (27):—

"I have had, up to the present time, the opportunity to examine four syphilitic soldiers, who suddenly became deaf after having taken cold. They suffered from vertiginous attacks, from which they recovered, but remained deaf in spite of all treatment. One of these patients died from typhus fever shortly after the beginning of his aural difficulty. On examination considerable hyperæmia was discovered in the mucous membrane of the drum, and in the membranous labyrinth, which appeared much thickened. The liquid within the labyrinth was sanguinolent and abundant." Gruber asks, "whether this condition is perhaps to be thought an inflammation with hemorrhagic exudation, to which syphilitic patients are predisposed? Further observations are needed."

That these conditions were of an inflammatory nature is evident. In regard to the *hemorrhagic* exudation, I must mention that hemorrhages in syphilitic inflammations of the inner membranes of the eye are of the rarest occurrence, while the abundance of the liquid and the hyperæmia in the labyrinth, found by Gruber, are in harmony with what we see with the ophthalmoscope under similar conditions in the eye. The hemorrhage in Gruber's case may, perhaps, rather be due to the typhus than to the syphilis of the patient.

For the sake of *recapitulation*, I deem it expedient to present a GENERAL SUMMARY of the principal points discussed in the foregoing paper.

When *deafness occurs almost suddenly, under apoplectiform symptoms*, in a previously healthy person, and the apoplectiform symptoms disappear rapidly and completely, but the deafness remains, then the seat of the disease is the cavity of the labyrinth, and its pathology probably hemorrhagic or serous effusion.

The deafness resulting from *injuries* on the head is caused by fissures or fractures of the petrous pyramid, with consequent hemorrhage and inflammation in the membranous labyrinth, or, perhaps, by isolated rupture and hemorrhage in the soft parts of the labyrinth, by "contrecoup."

The chain of symptoms known as *Ménière's disease*, viz., deafness and tinnitus aurium, occurring under symptoms of apoplectiform cerebral congestion, may be a *primary* or a *secondary* affection of the labyrinth.

The *former* is to be assumed when, in a previously healthy person, the symptoms on the part of the labyrinth —tinnitus, contraction of the range of audition, and hardness of hearing—are prominent and persistent, while the general symptoms—headache, paleness of the face, fainting, obscuration of the visual field, vertigo, nausea, vomiting, and disturbances of equilibrium—cannot be referred to actual disease of the brain or the constitution, and are transitory. The pathology of Ménière's disease is most probably hemorrhagic and serous inflammation within the whole labyrinthine cavity. *Increase of intra-auricular pressure* may be considered a primary consequence of it, upon which other symptoms—vertigo, nausea, vomiting, disturbance of equilibrium, etc.—depend. Glaucoma of the eye is, perhaps, a counterpart to Ménière's disease.

Ménière erred in confining the disease to the *semi-circular canals*, the accompanying deafness being evidence that the cochlea is involved. That the disease is neither an affection of the trunks of the auditory nerves, nor of their centre in the brain, is demonstrated by the deafness for high and low tones—a contraction of the range of audition—which I have witnessed. This symptom can be explained only by changes in those parts of the cochlea which are subservient to the perception of high and low tones. It is analogous to the contraction of the visual field in glaucoma.

Ménière's disease may be *secondary* to affections of the *tympanum*, the *brain*, and the *general system*. I have reported typical cases in proof of this assertion.

In a chronic *aural catarrh* the whole chain of the symptoms of Ménière's disease may occur as an acute complication—caused by exposure—disappear, and leave the chronic catarrh in its previous condition, but with increased deafness and tinnitus. In such cases the inflammatory symptoms of the labyrinth may be well marked without noticeable aggravation of the otitis media.

The deafness, mostly total, occurring in the course of acute *meningitis*, and sporadic or epidemic *cerebro-spinal meningitis*, is due to *purulent otitis interna*, a complication—perhaps a metastasis—of the purulent inflammation of the meninges, in the same way as purulent choroiditis is not an infrequent complication of the same diseases. Voltolini's opinion, that the whole affection in such cases is an inflammation of the labyrinth alone—otitis labyrinthica, mistaken for meningitis—lacks both foundation on pathological anatomy and clinical analogy.

Among the *general diseases* which are liable to produce deafness, *constitutional syphilis* furnished a valuable example in Case 42, showing a pure attack of Ménière's disease, in a patient who soon after had an analogous affection of his eyes. This being exudative irido-choroiditis, chiefly of a serous character, it may be inferred that the labyrinthine affection also was a serous exudative inflammation. *Severe fevers*—for instance, *typhus* and *typhoid; acute exanthemata:* variola, scarlatina, rubeola; *eclampsia infantum; erysipelas; pustular* and *eczematous eruptions on the head; mumps, angina*

Ludwigii, and *sunstroke* are all apt to produce deafness without material changes in the middle ear. The most probable anatomical lesion in all these cases seems to be serous or hemorrhagic inflammation in the labyrinth.

The lesion producing deafness after childbirth may be of the same nature or metastatic, as in severe puerperal fevers; anæmia, in certain cases.

We may, therefore, *subdivide otitis interna into three kinds:—*

1. *Hemorrhagic:* sudden and traumatic deafness;

2. *Serous exudative*, or *simple:* idiopathic Ménière's disease, and the deafness from syphilis, severe fevers, acute exanthemata, erysipelas, and other constitutional diseases.

3. *Purulent:* a *primary* disease of traumatic origin, perhaps also spontaneous and sui generis (Voltolini), and *secondary* in simple and cerebro-spinal meningitis, puerperal fever, and other pyæmic diseases.

The *diagnosis* of inflammation of the labyrinth is mainly supported by the symptoms of permanent irritation and paralysis of the auditory nerve:—tinnitus aurium and deafness—accompanied by transitory cerebral symptoms:—headache, paleness of the face, vertigo, fainting, nausea, vomiting, disturbance of equilibrium—without marked changes in the middle or outer ear. Secondary inflammation of the labyrinth is to be assumed when, in the general diseases enumerated above, tinnitus aurium and deafness, without symptoms of otitis media, are well marked.

The *prognosis* of idiopathic otitis interna varies with the nature and cause of the inflammation. In traumatic cases the hearing may be partially or almost totally restored. The secondary affections of the labyrinth in typhoid fever, syphilis, and other constitutional diseases may terminate favorably, or, on the other hand, leave all possible degrees of hardness of hearing, even total deafness being not infrequent. Idiopathic serous exudative otitis interna, if we may call Ménière's disease by that name, is mostly fatal to the hearing. If the first attack be not severe and do not destroy the hearing more or less completely, other attacks will follow at indefinite intervals, and gradually result in deafness. The course of this disease bears great analogy to glaucoma. Purulent otitis interna is almost always followed by complete deafness. Only in exceptional cases of cerebro-spinal meningitis hearing has been restored.[1]

The *treatment* of the labyrinthine affections is still more in its infancy than is its pathology. Antiphlogosis and derivation, both locally and constitutionally, with due regard to the nature of the general symptoms, are indicated in all recent cases. Leeches behind the ears, aperient remedies, among which calomel may be preferred; sudorifics: digitalis, etc., and absorbents: hydrargyrum, iodide of potassium, etc.; afterward vesicants over the mastoid region should be recommended. Electricity, according to different methods, has nearly always failed; Brenner's method, however, claiming success in exceptional cases, may be tried. I have employed it in

nearly all the reported cases, but without a shade of improvement. If the increase of intra-auricular tension were better known as a fundamental and detrimental symptom in certain inflammations of the inner ear, we should endeavor to get access to the cavity of the labyrinth with proper instruments, perhaps best by one of the fenestræ, in order to evacuate a part of the perilymph.

LITERATURE ON THE SUBJECT.

(The papers marked with * have been read by me in the original, the others in extracts.)

*1. Paul Ménière, *Gaz. Méd. de Paris*, 1861, pages 29, 55, 88, 239, 379, 597.

*2. Von Troeltsch, *Treatise.* Lecture 28th.

*3 A. Politzer, Lesion of the Labyrinth. *Arch. f. Ohrenh.* II. pp. 88–99. 1865.

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TWO CASES OF SYMPATHETIC OPHTHALMIA DISTINGUISHED BY THE OCCURRENCE OF NEURO-RETINITIS.

By THOS. R. POOLEY, M.D., of New York,

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Amongst the complex array of symptoms which manifest themselves in sympathetic affections of the eye, lesions of the retina have not been described. Mooren, in his valuable monograph on this subject, does not mention neuro-retinitis as coming under his notice. I have lately seen two cases of this form of disease in eyes sympathetically affected, and have thought that it might not be uninteresting to bring them before the notice of the profession.

The first case was that of a boy, aged 9 years. He came under my care July 27th, 1869. The preceding day he was struck upon the left eye by a clam-shell, with considerable force. There was a wound on the lower and inner sclero-corneal margin, about 3‴ in extent, 2‴ of which were in the cornea and 1‴ in the sclerotic. The iris was engaged in the wound, and covered by adventitious tissue. Hemorrhage into the anterior chamber obscured the lens, and prevented illumination of the fundus; vision reduced to quantitative perception of light. Atropine

was ordered, and a compress bandage applied. The following day the blood had been so much absorbed that he could count fingers at 2′. The eye continued to improve, the blood all became absorbed, and vision increased to $\frac{2}{3}$. August 24th I first noticed symptoms of irritation of the other eye. I did not see him again until the 28th, four days later. The injured eye was now hyperæmic, painful upon pressure, and by reflected light an exudation behind the lens, in the immediate location of the wound, was distinctly to be seen. The right eye was injected, the pupil sluggish, and under atropine showed a synechia towards the nasal side.

The following day, with the concurrence of Dr. Knapp, who was asked in consultation, I enucleated the injured eye. The operation was followed by a temporary amelioration of the sympathetic affection; but it afterwards progressed, running the usual course of irido-choroiditis, until October 11th, when the symptoms for which this case is reported declared themselves. At this time, although the vitreous was hazy, a pretty distinct view of the fundus could be obtained, the retinal vessels were full and tortuous; the optic disc covered by exudation, so as to obscure its border and vessels. Some days later the refractive media became too obscure to admit of a view of the fundus, and therefore the course of the neuro-retinitis could not be followed. The sympathetic inflammation now steadily progressed, in spite of all treatment. The iris became attached to the lens capsule, the vitreous still more cloudy, exudation into the pupil took place, the anterior chamber decreased in depth, and exudation also occurred on the posterior surface of the cornea. Vision was reduced to $\frac{5}{200}$.

When last seen, a few months later, the appearance of the eye was substantially the same as above described, except that all active inflammation had disappeared, and he still retained the same degree of vision.

ANATOMICAL EXAMINATION OF EYE-BALL.

Macroscopical Appearances.—The eye had lain in Müller's fluid for six weeks. Antero-posterior diameter

of globe, 23 mm.; transverse, 23½ mm. The inferior part of the globe is distended. Meridional sections show all the membranes attached, except the choroid, which is detached; there is, however, no fluid between it and the sclerotic, conditions which show the detachment to be due to the opening of the globe. The scar commences at the median line, on the lower portion of the globe, extends 8½ mm. in a direction upward, inward, and backward from the sclero-corneal margin through the ciliary body. The vitreous coagulated like jelly, by the effects of the hardening fluid, detaches readily from the retina in the posterior portion of the globe, but is inseparably united to it in the anterior and ciliary regions, the union being densest at the place of the scar.

Microscopical Examination.—In the posterior portion of the vitreous, lymphoid and larger irregular cells were scantily scattered; moreover, a net of very pale, large stellate cells, with many anastomosing offsets, were discernible; more anteriorly the same elements increased in number, and became more distinct (less pale).—*Retina:* Sections of the posterior portion showed all layers perfectly normal. Further sections showed that the retina continued normal throughout its entire extent. At the ora serrata the intumescence, which the retina shows in its normal condition, was increased. Its ciliary region was very much enlarged, and thickly strewn with lymphoid cells. The fibres of the zonula Zinnii were very distinct, and likewise densely crowded with lymphoid bodies, without any new-formed blood-vessels. Fila-

ments, connective-tissue cells, and lymphoid cells extended from the zonula into the vitreous, behind the lens.—*Choroid:* Shows throughout its whole extent circumscribed foci of infiltration with lymphoid bodies, which were more or less densely imbedded in the interstitial spaces of the choroidal stroma, leaving the white as well as the colored stellate cells unchanged. The infiltrations had only taken place in the vascular and capillary layers of the choroid, swelling them to about double their normal thickness, while the hyaline and pigmentary layers were quite intact.

There was no visible change in the walls or contents of the choroidal blood-vessels (indicating phlebitis, thrombosis, or other changes). The choroid readily detached from the retina and sclerotic. The loose cellular tissue between it and the latter showed the usual sparse net of large anastomosing stellate cells, with here and there a scanty amount of lymphoid bodies scattered among them.

The *Iris* presented throughout its whole extent a thick and uniform infiltration with lymphoid bodies, without alteration of its blood-vessels and pigment layer. The same infiltration also existed in the ciliary processes. The pigment layer of the latter was lacerated in some places; the cells strown out into the proliferating ciliary part of the retina, as has been above described. The ciliary muscle presented no abnormality whatever; the cornea was quite normal. *Transverse sections through the region of the wound showed the posterior portion of*

the iris and the anterior portion of the ciliary processes caught within the scar, and their elements compressed and distorted. In the closest neighborhood of the scar, the cornea, sclerotic, and ciliary muscle proved normal, while the iris, ciliary processes, and choroid exhibited the inflammatory processes described.

A *ciliary nerve* taken from behind the scar, and placed under the microscope, did not exhibit any abnormality of structure, except some very few lymphoid cells which were seen between the fibres.

The above conditions may be *summed up* as follows:— A perforating wound at the corneo-scleral juncture gave rise to implication of the iris and ciliary processes in the cicatricial tissue, and chronic exudative irido-choroiditis of partly plastic, partly purulent character.

A direct cause of production of sympathetic irritation of the other eye could not be anatomically demonstrated, and must be sought for in the physiological connection which exists between both eyes. If we endeavor to explain it by nervous action, we may assume that the nerves of the iris caught in the wound were compressed by the contraction of cicatrized tissue, and reflected the irritation to the other eye.

Case II. The patient, a man æt. 59, was brought to the Ophthalmic and Aural Institute, Oct. 20th, 1870, by Dr. Elting, of Tremont. When he was 19 years old, his left eye was injured by a wire; it became severely inflamed. The inflammation subsided, but the eye remained subject to attacks of the same character whenever he was exposed, or caught cold. By these repeated attacks it became increased in size,

and bulging, more especially in the outer anterior sclerotic region. Dr. Elting stated that about five months ago the eye was nearly double the normal size, showed a spherical corneal, and an irregular ciliary and scleral staphyloma. The beginning of last June he injured the eye again, by striking it against a stick while bending his head forward. Following this injury the eye became inflamed, more bulging on its outside, perforated, showed a flesh excrescence, and then gradually shrunk. After this occurrence it became perfectly painless for two or three months. Three weeks before his admission his right (the other) eye diminished in sight, and a fog appeared before it. He called on Dr. E., who found the eye-ball tender, slightly injected, and the sight impaired. Antiphlogistic treatment was resorted to, the tenderness disappeared, but the injection and diminution of sight remained. The doctor considered the inflammation of a sympathetic nature, and therefore brought him to Dr Knapp for advice.

On admission his condition was as follows:—*Left eye*, sight œdematous, swelling of lids, marked conjunctival and sub-conjunctival injection, cornea very much shrunken, sclerotic wrinkled. The eye-ball much reduced in size, and hard to the touch, almost like bone, movements impeded in all directions; there was tenderness and lachrymation. In the other (*right*) *eye*, vision reduced to counting fingers at 5′; shallow anterior chamber, dull pupil, discolored iris, numerous synechia, some of which were torn by the use of atropine; vitreous cloudy; retina seen as if through a veil, but sufficiently well to distinguish a high degree of hyperæmia of the optic disc and vicinity; uniform and semi-transparent exudation on and around the disc, the borders of which were completely concealed.

The injured eye was *enucleated* forthwith, and the patient remained under treatment in the Institution for six days; at the expiration of which time his iritis had disappeared, the synechiæ were all torn, and his pupil was widely dilated. The optic nerve was still swollen, but the exudation had considerably diminished. S = $\frac{20}{200}$. Nov. 2d, he came to the clinic. Exudation was still less, and S increased to $\frac{20}{100}$. —Dec. 19th the neuro-retinitis had almost entirely disappeared, and

S = $\frac{20}{40}$.—April 1st. Had been free from unpleasant symptoms; until lately his eye was somewhat sensitive to the light. The vessels of the papilla were found more dilated and tortuous than before. Leeches on the temple, and an aperient, were ordered.—April 14th. The eye had recovered its usual strength, V = $\frac{20}{30}$. A low degree of hyperæmia of the disk and surrounding retina still existed; indeed, this hyperæmia never had completely disappeared as long as the patient was under observation.

Anatomical Examination of Eye-ball—Macroscopical Appearances.—Eye hardened in alcohol, much shrunken. An antero-posterior section, passing through the centre of the cornea and optic nerve, discovers the following conditions: Sclerotic slightly thickened on its inner side —but very considerably—to about four times its ordinary thickness on the outer. This thickening is, however, more apparent than real, since it encloses a cavity filled with a yellowish grumous substance, which is surrounded by a soft fibrous tissue. The cornea is reduced in size to about $\frac{1}{3}$ of its natural diameter. The anterior portion of the globe represents a total disorganization of its natural parts: the lens, iris, and ciliary body having disappeared, and their places, as well as the anterior and posterior chambers, being entirely occupied by dense fibrous tissue. In connection with it is a transverse septum, which contracts into a cord-like prolongation, running through the centre of the vitreous chamber and inserting itself with a tapering end at the optic nerve. The space between it and the sclerotic is partly empty, partly filled with a grayish substance, and lined at its periphery with a fibrous coat.

Microscopical Examination.—The corneal substance still recognizable as such, but its lamellæ and corpuscles irregularly arranged and distorted, resembling undulating fibrous tissue. No epithelium visible. In the outer layers of the cornea, yellowish, semi-transparent, globular, and elongated masses are imbedded in clusters or undulating figures. They had a colloid appearance, and remained unchanged when treated with muriatic, nitric, and acetic acids. The majority of them are in connection with the inner surface of the thickened anterior elastic (Bowman's) membrane; some of them appear disconnected from it in the anterior strata of the cornea. They represent a colloid degeneration of the anterior elastic membrane and outer lamellæ of the cornea, first described by Dr. H. Althof.

Behind the corneal substance lay irregularly arranged connective tissue, and behind this the lens capsule greatly wrinkled, but unchanged in structure. It encloses a grumous, softish mass—the broken-up lens substance. Immediately behind lay the remnants of the disorganized iris and ciliary body—the ciliary processes, although distorted, still recognizable by their abundance of pigment. The transverse fibrous septum is made up of a finely striated matrix, in which, here and there, quantities of small round cells are imbedded; in some places yellow amorphous pigment is scattered in irregular masses, among which well preserved or ragged blood corpuscles are to be seen, demonstrating its derivation from blood. The cord-like prolongation of the transverse

septum manifests itelf as the retina, entirely changed into connective tissue, to the periphery of which, in the anterior parts, the outer layers of the ciliary processes are adherent, while in its posterior parts only choroidal pigment is irregularly scattered through the outer layer. The fibrous membrane lining the inner surface of the sclerotic was the choroid, the outer layers of which were changed into connective tissue, with regular striated arrangement of the fibres; but the pigment layer was thickened, or rather converted into an irregular stratum of small round cells or granules, imbedded in a finely dotted matrix and interspersed with choroidal pigment. These changes are to be considered as the result of a chronic inflammation producing new-formed connective tissues in the outer, and pus in and upon the inner layers of the choroid. The grumous substance in the cavity enclosed by the sclerotic consisted of fat in molecules and small lobules. The soft fibrous tissue around it was loose connective tissue, showing large anastomosing stellate cells in a homogeneous matrix resembling mucous tissue, which is very prone to form smaller and larger cystic spaces. No nerves were found in the disorganized tissues, but it is probable enough that the ramifications of the ciliary nerves in the ciliary muscle and iris were compressed, altered, and irritated by the destructive inflammation and shrinking of these parts. It seems the most natural to seek in this condition of the nerves the starting-point of the sympathetic affection of the other eye.

ON VERTIGO OCCURRING IN AFFECTIONS OF THE EAR (AUDITORY VERTIGO).

By Dr. GUSTAV BRUNNER, of Zurich.

Translated by Dr. Emil Gruening, of New York.

Every aurist is aware that vertigo occurs in various morbid conditions of the ear. This remarkable connection presents many points of interest, and I therefore thought it appropriate to collect all the facts bearing upon the subject. Before discussing the topic I may be allowed to make some remarks on vertigo in general.

We may briefly define vertigo as an impairment of the sense of equilibrium.* It is by no means essential that the equilibrium itself should in reality be disturbed,

* In the present state of our knowledge it is difficult to give an accurate and comprehensive definition of vertigo. Hasse (Diseases of the Nervous System) says: "The connection between the sensory and motory excitation of the brain seems to be deranged in such a manner as to lead to the conception of apparent movements, either of the body or of external objects, and to produce involuntary movements in accordance with this delusion, falling, and loss of consciousness."

or even imperilled. It suffices that one be under the impression, well grounded or not, that the equilibrium is or may become deranged. This fact is illustrated by the sensation of vertigo which we have when looking into a great depth.

We are justified in supposing that there exists in the brain a centre of equilibrium (it is unknown where) which regulates the balance of the body, and executes at every instant such combined movements as are requisite for the preservation of the centre of gravity. These movements are performed instinctively, without the intervention of the intellect or without our consciousness. For this purpose the centre must not only obtain from our senses accurate information as to the relative positions of the body, but also be suitably connected with the centres of motory co-ordination. The task of this centre to respond to impressions conveyed by the senses, and to send forth motory impulses, constitutes a psychical function. In order to obtain a thorough insight into the mechanism of vertigo, it is requisite to possess an accurate knowledge of the component parts of this apparatus, its afferent and efferent nerves, and the manner of their communication. The supposition that alterations in one of these factors may occasion dizziness is borne out by experience. For our special task it is of importance to know in what manner the auditory nerve is connected with the apparatus of equilibrium.

Of the means of information we may mention first

the muscular sense, the nature of which is not yet fully understood; secondly, the senses of sight and touch. It is well known that the muscular sense plays an important part accessory to the functions of the eye in forming our ideas of space. This is proved by the occurrence of violent vertigo in paralysis of the ocular muscles. Nothing definite can be stated as to the degree in which the sense of hearing participates in forming our ideas of space. I am of opinion that in this it has no large share.

Vertigo is generally classified as sensuous, symptomatic, and sympathetic, to which we may add psychical and toxic vertigo.

Sensuous vertigo is produced by violent impressions on the senses which the mind is incapable of harmonizing with the actual state of equilibrium, maintained instinctively by the muscular sense and the sense of touch.

Hasse says: "As our consciousness is not accustomed to resolve rapidly and accurately violent impressions, perception becomes confused, and disproportionate motory innervation, in brief, vertigo ensues."* Most commonly sensuous vertigo is occasioned by visual impressions. Whether impressions upon the other senses, especially the sense of hearing, produce a similar result, has not yet been elucidated. I am inclined to doubt it. Hasse, after discussing vertigo dependent upon visual impres-

* This explanation does not seem to agree with the fact that children, though less accustomed to resolve unusual sensuous impressions than adults, suffer less from vertigo (*i.e.*, in turning).

sions, says: "Cases are rare in which vertigo is caused through the agency of the sense of hearing." He cites no cases. There are, however, cases on record in which vertigo occurred in consequence of auditory impressions,* but in those cases the relation of cause and effect seems to differ from that same relation in visual dizziness. It is the intense impression of sound as such which [perhaps as violent fright] produces vertigo, *i. e.*, an incongruity of the auditory impressions on the one side and the muscular sense and visual conceptions on the other.

Vertigo dependent upon affections of the central nervous organs is called *symptomatic.*

The greatest obscurity prevails as to those cases which are designated as *sympathetic* vertigo. This occurs in affections of the stomach, of the liver, and of the kidneys, in helminthiasis, and in anomalies of menstruation. Though recent investigators have, with success, endeavored to limit the domain of sympathetic vertigo by considering gastric and renal vertigo as toxic, and cardiac vertigo as dependent upon disturbances of circulation, there still remain numerous cases in which these interpretations prove unsatisfactory. I do not believe that gastric vertigo can always be traced to a toxic origin. Observations have been made which seem to demonstrate that the pneumogastric nerve has a special bearing upon the occurrence of vertigo; it is as yet im-

*Schmidekam observed such a case in his own person.

possible to conceive what influence this nerve could exert upon the mechanism of equilibrium. If we endeavor to explain it by its relation to the vascular system, we are unable to account for the fact that vertigo is constantly attended with nausea and vomiting. We might also suppose that the cause could be found in either an accidental contiguity or a still unknown but essential communication of the pneumogastric nerve with the centres of equilibrium.

We shall now consider vertigo taking its origin from the ear.

Vertigo may be caused by external pressure upon the drum-head, by the impulsion of air or liquids into the tympanic cavity through the Eustachian tubes, or by the passage of a galvanic current through the ears. It also occurs in pathological conditions of the midde ear or of the labyrinth, especially in Ménière's disease, and in otitis labyrinthica of Voltolini. Whether morbid changes of the auditory nerve or its roots may also produce vertigo, we do not yet know.

I shall first treat of those cases in which vertigo is produced by the impulsion of air or liquids into the tympanic cavity, that is, by blowing the nose, or by Valsalva's, Politzer's, and Gruber's methods, or by the application of the catheter for air and liquids. All these methods produce an increase of pressure within the tympanic cavity.

In Valsalva's method, the air entering the tympanic cavity will exert an equal pressure in all directions, and

force the drum-head outwards, and the membranes of the fenestræ inwards. This supposes the correctness of the view of Helmholtz, that only in inward movements the handle of the hammer carries with it the anvil, while in outward excursions the ratchet-teeth of the malleo-incudal articulation are disengaged. Therefrom will necessarily result a pressure upon all the soft parts within and surrounding the fluid of the labyrinth, and thus also upon the terminal expansion of the auditory nerve. Owing to various circumstances, vertigo is by no means a constant result of the methods mentioned. I frequently made the observation, that at a certain phase of treatment the impulsion of air, either by Valsalva's method or by merely blowing the nose, will produce momentary vertigo which did not occur previously. According to my view, this may principally be due to the fact that a partial obstruction of the Eustachian tube acts like a valve, admitting air under a certain pressure, but preventing its exit. It may also occur whenever, after complete closure of the Eustachian tube, the drum-head remained curved inwards for a certain length of time, and then by the sudden introduction of air is forced outward so much the more strongly. In the course of treatment the acoustic nerve at times becomes irritable, and produces a tendency to vertigo. It is undoubtedly true that, in cases of anchylosis of the malleo-incudal joint, the stapes is somewhat lifted out from the fenestra ovalis by Valsalva's method, and a different effect is produced. It is highly probable, but

not conclusively proved, that in all the methods mentioned, vertigo is occasioned by the increase of intra-auricular pressure; other circumstances may, however, concur to produce this symptom.

External pressure upon the membrana tympani does not seem to cause vertigo as readily, since it differs in its effects from Valsalva's method, and resembles a normal impression. Here pressure is exerted upon one fenestra, causing the fluid of the labyrinth to move toward the other. I am well aware that vertigo often occurs in syringing ears whose drum-heads are perforated. I am at present treating a lady suffering from chronic catarrh of the middle ear without perforation, in whom vertigo can be induced more readily by this method than by Gruber's. But this is by no means the rule. It is known that in cases of defective membrane and suppuration in the tympanic cavity the syringe must be used very cautiously. I am inclined to think that in such cases not only the defect of the membrana tympani ought to be taken into consideration, but also the hyperæmia and increased irritability of the labyrinth, for in my experience these accidents most frequently happen in neglected cases of profuse otorrhœa dependent upon hyperæmia and granulations of the tympanic mucous membrane, and probably upon caries.

When Schmidekam * brought the pressure of a column of cold water of 54 to 117 centimetres height

* Experimental researches.

to 'bear' upon his tympanic membranes by means of a rubber tube, he felt intense pain in his ears, and suffered from vertigo and nausea amounting almost to syncope. He was compelled to lie down, whereupon retching and vomiting ensued. When he repeated this experiment with a column of water of the same height, but of a temperature of 25° Réaumur, none of these unpleasant symptoms occurred. The latter may be explained by the irritation of the nerv. auricul. vagi with cold water. Irritation of this nerve often produces strange phenomena.*

It is surprising that Magnus,† in his interesting experiments instituted at the building of a bridge in Königsberg, under a pressure of 2½ atmospheres, does not mention vertigo as one of the symptoms. In the majority of cases he observed buzzing noises, hardness of hearing, and violent pain. These symptoms ceased whenever the pressure on both sides of the tympanic membrane was equalized by either a swallowing movement ‡ or Valsalva's method.

* The cold water may have caused a considerable hyperæmia of the inner ear.

† Archiv f. Ohrenheilkunde, I., page 269.

‡ Supposing that in the persons experimented upon the Eustachian tube was normal, we have no proof of the non-existence of a small air-passage beneath the hook-like cartilage. We can readily conceive that such a passage might not have sufficed to equalize the considerable difference of pressure. A swallowing movement was therefore required.

The pain was evidently due to the great tension of the membrane. In all the cases the latter had a scarlet-red color, an abnormal reflex, and was strongly curved inwards. Immediately after the equalization of the pressure all the unpleasant sensations disappeared, and Magnus states that the impulsion of air through the tubes was the only measure required to guard against every inconvenience.

In all these cases there existed a pressure of $2\frac{1}{2}$ atmospheres upon both fenestræ of the labyrinth, *i. e.*, an excess of pressure of $1\frac{1}{2}$ atmospheres. After Valsalva, the excess of pressure can only amount to $\frac{1}{8}$ of an atmosphere, as a vigorous man blowing with the full force of his lungs into a manometer will hardly, as I have seen myself, overcome more than the pressure of $\frac{1}{9}$ of an atmosphere. The pressure in the pneumatic cabinet of Lewinstein amounts to $\frac{1}{5}$ to $\frac{2}{5}$ of an atmosphere. It is a mere supposition, but in analogy with the result obtained by galvanic irritation, that vertigo is less readily occasioned by simultaneous pressure upon both ears.

Very recently Friedrich Eugen Weber, of Berlin, found * that a colored fluid injected into the arachnoidal space follows the course of the lymphatics through the aquæductus cochleæ into the scala tympani. These experiments, if confirmed, will show that there exists a close relation between the fluid of the labyrinth and the cerebro-spinal fluid.

* Monatsschrift f. Ohrenheilkunde, 1868, page 105.

I do not, however, think it advisable to draw our conclusions from these experiments.

Other methods, by which either air or liquids are forced into the cavity of the tympanum, produce effects similar to those of Valsalva. It requires no lengthy explanation to prove that in all these cases real vertigo ensued, and not a condition resembling syncope, as Dr. Benedict maintained in my presence. We find no disturbance of consciousness, no pallor of the face, no feebleness in the action of the heart, and the patient does not only describe his condition as vertigo, but we are also able to determine this objectively by the unsteady and tottering movements, which show themselves especially in rising from a seat and in walking. Such patients will therefore remain in their seats till the unpleasant sensation of vertigo disappears. Many patients are able to state towards which side they stagger, and it would be interesting to know whether this corresponds to the side of the affected ear, as after application of the galvanic current.

In 1862 I observed a very interesting case, which seems to prove that variations of intra-auricular pressure react upon the motory centres.

A vigorous country lad, aged 14, whom I treated for chronic aural catarrh, was suddenly, during the application of the *air-douche*, attacked with left *hemiplegia* and violent *vertigo*, without, however, losing his consciousness. The hemiplegia disappeared after a quarter of an hour, and the patient was able to walk to his home, at a distance of at least 2 miles. The paralysis occurred suddenly, without the least pain; the patient only feeling dull and dizzy. I cannot state positively whether

the hemiplegia occurred on the side to which the catheter was applied or on the opposite, but I am under the impression that the former was the case. Whether sensibility was impaired, or reflex movements could be induced, I do not know. I was, at the time, somewhat perplexed at the suddenness of the accident, and omitted to make a thorough examination. I remember, however, that voluntary mobility was completely abolished in the left upper and lower extremities and in the facial muscles of one side. This condition lasted about 10 minutes, after which time the patient gradually regained control over his muscles. He stated that the muscles of the toes and fingers were restored first.

I may add that the patient had always enjoyed good health, and never suffered from nervous symptoms. Previous to the accident I had employed the air-douche for a long time, and I subsequently continued its use, with due precaution, without its giving rise to any unpleasant symptoms.

The hearing of the patient was not affected by the accident, and the treatment of the chronic aural catarrh yielded, upon the whole, a favorable result.

Before endeavoring to explain these phenomena, I shall speak of the manner in which the air-douche was applied. I used at the time Rau's compression pump, but with an enlarged receiver. This was charged by 40 strokes of the piston. Previously I had often charged it by 60 to 70 strokes. The former charge corresponds to a pressure of $\frac{1}{3}$ of an atmosphere, or 5 pounds; the latter to $\frac{1}{2}$ of an atmosphere, or $7\frac{1}{2}$ pounds. A large rubber bag, gently compressed, yields a pressure of 2 to $2\frac{1}{2}$ pounds, which, by vigorous compression, may be raised to 4 and $4\frac{1}{2}$ pounds. In order to obtain such results of pressure the receiver or bag must be hermetically connected with the manometer.

The conditions are changed when the catheter is but loosely inserted into the ostium pharyngeum, and the impelled air partly escapes. In this case a certain degree of tension of the air-receiver may produce various degrees of pressure within the tympanic cavity, according to the more or less intimate connection of the catheter with the Eusta-

chian tube, the caliber of the catheter, and the position of the faucet for the regulation of the entrance of air. When I charged the receiver, as stated above, with 40 strokes of the piston, and inserted the catheter but loosely into the manometer-tube, the pressure of 5 pounds commonly obtained by the hermetic connection was reduced to $\frac{1}{2}$ pound, and with the faucet partially closed to $\frac{1}{30}$ pound. We thus see that the degree of pressure depends chiefly upon the position of the catheter. The use of the compression-pump requires careful attention. At present I generally employ the air-bag, and but occasionally the pump, for diagnostic purposes.

How could we explain the occurrence of that remarkable accident?

No one will assume that it was caused by an emphysema of the ostium pharyngeum tubæ. The introduction of the catheter was very easy, and the symptoms did not justify the diagnosis of so rare an occurrence. The fact that the hearing was not impaired by the accident proves that a serious trouble, *e. g.*, laceration of the membranes of the fenestræ with escape of the fluid of the labyrinth, did not occur. It is well known that the roof of the tympanic cavity is at times defective, and in one case Gruber, after forcibly injecting a corpse, found the dura mater covering the defect considerably raised. But in this location a projection inward of the dura mater could not have caused hemiplegia. Furthermore, the complete absence of subsequent symptoms and the improbability of the existence of such a defect in so young a person strongly militate against this supposition. It is highly probable that the sudden increase of intra-auricular pressure, which under certain conditions pro-

duces phenomena of vertigo, gave rise, in this case, to a reflex neurosis.

In a man whom I treated for chronic aural and tubal catarrh with obstruction of the Eustachian tubes, I once observed, during the impulsion of air by means of the catheter and compression-pump, an involuntary trembling of both upper and lower extremities, violent dizziness, and slight stupor. Consciousness was not abolished. All these phenomena disappeared after a few minutes. This very patient, who was subject to occasional attacks of vertigo, made the observation that, even without the introduction of air, vertigo ensued whenever the beak of the catheter was suddenly rotated in a certain direction. I imagined that this manœuvre rendered the calibre of the Eustachian tube gaping, and suddenly produced a change in the intra-auricular pressure.

Of the two cases of death which occurred in the practice of Dr. TURNBULL, in consequence of catheterization of the tubes, one may probably be explained in that manner. The patient, a boy, aged eight, fell back in his chair during the operation and never recovered. If we assume that vertigo, syncope, hemiplegia are occasioned by reflex action, may we not also suppose that momentary paralysis of the great centres of vegetative life are caused in the same way? The possibility of this occurrence will be readily conceived by considering that the auditory nerve takes its origin in the medulla oblongata, which constitutes the centre of the respiratory and the combined reflex movements.

I shall now treat of vertigo produced by galvanization. Hasse states, in his treatise on affections of the nervous system, that, according to Purkinje, vertigo is occasioned

by the passage of a galvanic current through the brain from one ear to the other. Very recently Brenner published his interesting observations on this subject.*

Vertigo constitutes, according to Brenner, one of the most annoying accessory effects in the galvanization of the ear. This symptom requires the most careful attention of the electro-therapeutist, as the consequences to which it may lead, when occurring with a certain degree of intensity, are as yet unknown. Brenner illustrated the oscillations of the equilibrium by fastening a pencil to the head of the person with whom he experimented, and showed that the person may fall and even swoon. In order to produce vertigo, it is requisite that the line connecting the two electrodes should be parallel to the sagittal suture, *i. e.*, that the poles should be applied to the same side of the cranium. The most intense effect will be obtained whenever the direction of the current is at right angles with the sagittal suture. The cranial regions to which the poles may be applied differ in significance. Vertigo is produced with great difficulty by applying the poles to the lower portions of the face, but very readily by passing the current through the nape of the neck or the mastoid processes. Immediately after the closure of the current the person experimented upon bends his head and trunk to one side, and remains in this position till the current is opened, whereupon he resumes his former attitude as if he were forced back by an invisible hand. When the attack of vertigo passes away, the patient complains of intense nausea, and feels a dull and oftentimes prolonged pain in the center of the occiput over the protuberance. The loss of equilibrium ensues without exception towards the side of the anode. It can be demonstrated objectively that the sensation of falling towards one side is no delusion. The appreciation of the degree of inclination, however, becomes exaggerated. If the experiment is made with the eyes closed, even a weak current will suffice to produce the sensation of the head describing an arc of a circle of about one foot. On opening the eyes

* Researches and Observations on Electro-Therapeutics. Vol. I., Part I., pp. 75–78.

the small extent of the movement will be readily recognized. When the cathode is placed upon the nape of the neck, or any part of the trunk or limbs, and by doubling the other conducting wire two anodes are formed, one of which is applied to the mastoid process of one side, violent vertigo with inclination toward that side ensues at the closure of the current. As soon as the other anode is placed upon the opposite mastoid process, all symptoms of vertigo disappear. Therefrom we may infer that vertigo is produced by the application of the anode to one side of the head, but is not produced by the simultaneous action of the anode upon both sides of the head. According to Brenner, vertigo cannot be occasioned by induced currents.

These experiments are in many respects interesting, but permit a merely negative inference as to the part the organ of hearing plays in the production of vertigo. By placing the poles in a certain manner (in the vertical antero-posterior plane) it is possible to excite the auditory nerve without producing vertigo. As unavoidable accessory phenomena in the galvanization of the ear, Brenner mentions merely the reaction of the sensitive fibres of the fifth pair and the contraction of the muscles supplied by the facial nerve. Among the other accessory phenomena vertigo holds the second rank as to the frequency of its occurrence. Contraction of the masseter muscles is more frequent, and sensation of light ranks next to vertigo. Thus these experiments lead us to infer that the excitation of the terminal apparatus of the auditory nerve, by increased intra-auricular pressure, proves insufficient to produce vertigo.

We may here mention the following observation made

by Schmidekam.* He conducted the strong sound of a sirene through a caoutchouc tube directly into his ears, whereupon he suffered from slight vertigo, moderate nausea, tinnitus aurium, and subsequently from oppression.

We shall now treat of the various morbid conditions of the ear which may produce vertigo.

If we commence externally, we must first mention such phenomena as are caused by the presence of foreign bodies in the meatus externus. It is well known that impacted cerumen gives rise to vertigo, which is generally accounted for by the pressure upon the membrana tympani and the ossicles. The presence of foreign bodies in the meatus externus gives rise to various symptoms, which Moos, in his "Clinic of Diseases of the Ear," considers as reflex phenomena, and specifies as follows: epilepsy, hemiplegia, convulsions, unilateral anæsthesia, vomiting. It is surprising that Moos does not mention vertigo. But we may suppose that this symptom was sometimes overlooked. When the foreign body was removed the symptoms either disappeared or abated. In many of the cases mentioned the examination was not sufficiently thorough. In others all the phenomena were caused by the presence of insects in the auditory canal. This proves that increase of intra-auricular pressure ought not to be considered as the only cause.

* L. c., page 49.

At all events, the supposition of the existence of an increase of intra-auricular pressure, especially in such cases as exhibit these symptoms for a certain length of time, can only be maintained with due precaution. We can readily conceive that foreign bodies may produce inflammation either by their wedge-like pressure or by their sharp edges. The inflammatory process may be propagated into the labyrinth, and give rise to all the symptoms mentioned. A gradual cessation of these symptoms is an indication of this course.

Before discussing the affections of the middle ear, I shall speak of the diseases of the labyrinth in their relation to vertigo.

Our knowledge of the diseases of the inner ear is as yet very defective, especially as to primary affections of the labyrinth. What we know is derived from pathological conditions accidentally discovered post-mortem. But it seems that vertigo is a principal and constant symptom.

Two groups of symptoms, viz., the so-called Ménière's disease and otitis labyrinthica of Voltolini have recently been observed and described. They are said to depend upon a primary affection of the labyrinth. A series of reasons may be cited indicative of a connection of both these affections with the inner apparatus of hearing, though by pathological research nothing definite has as yet been demonstrated.

Voltolini,* who had not read Ménière's original paper, supposed that the symptoms occurring in both affections are identical. But I must here remark that this is not

* Monatsschrift f. Ohrenheilkunde, 1870, page 107.

the case, and accordingly, the anatomical changes must differ. Ménière's disease occurs without fever, as an attack of violent vertigo, often causing a fall. It was previouly considered as apoplectiform cerebral congestion. It has many points of resemblance with epileptic convulsions, and is principally observed in adults. Voltolini's disease, on the contrary, resembles meningitis, and is even considered as such by his opponents. It is a febrile affection, having an average duration of 8 to 10 days, and attacking children *par excellence.*

I may be allowed to discuss these points more in detail, as they are of importance for our theme.

To Menière is due the merit of having first indicated a group of symptoms, which previously had been considered as apoplectiform cerebral congestion, and treated, according to the records of the cases, by heroic blood-letting. In the year 1861 Ménière read before the Acad. de Médecine a paper* entitled: "Sur des lésions de l'oreille interne donnant lieu a des symptômes de congestions cérébrale apoplectiforme." At the same time he published in the "Gazette Médicale" a series of observations on this subject, and described the disease as follows:—

A person in good health is suddenly, and without an assignable cause, attacked with vertigo, nausea, and vomiting. His face becomes pale, bathed in perspiration, and indicates the approach of syncope. At times the patient succeeds in maintaining the erect posture, or in seating himself; but often he falls to the ground as if stricken with apo-

* Gazette Médic. de Paris, 1861, page 597.

plexy (one might say, "stricken with vertigo"). Consciousness remains often intact; in violent cases it vanishes temporarily during the paroxysm, but reappears speedily. The patient is unable to rise; every attempt to move is attended with intense nausea and vomiting. He clings to his couch for fear of being thrown off. He has the sensation of being rocked by the waves; all objects seem to whirl around him. His muscles are in a state of relaxation; only once I observed twitching of the muscles of the face, with subsequent hemiparesis (on the side of the affected ear) of several days' duration. The patient, a physician, retained his consciousness during the attack. In a second case, the patient, also a physician, noticed that in walking and standing he reeled towards the side of the worse ear, as if that side were not as fully under the control of the will as the other. When lying on his back he could move either side with equal ease. These phenomena disappear gradually in a very short time, often after several days, but recur sooner or later, at times within a month, at times after years. In the intervals the patient enjoys good health. At times a tendency to vertigo persists after the attack, the patient becoming vertiginous after rapid motions of the head, and showing great unsteadiness in the carriage of the body.

The attacks may never recur in subsequent years, and the general health of the patient may remain unimpaired. Ménière never saw epilepsy develop after the recurrence of the attack. He observed the disease, not only in adults, but also in children. He lays great stress upon the impairment of hearing, which constantly ensues with more or less rapidity, either in one ear or in both ears. In several instances the attacks were preceded by noises which continued during the intervals, and increased with every new attack. He found that in several cases the previously normal hearing diminished very rapidly after the attack. The hardness of hearing defied every means of treatment, and the objective examination constantly yielded a negative result.

The following two cases reported by Ménière may serve as examples:—

A highly educated lady, who had excellent hearing, and was personally known to Ménière, was one afternoon, after her usual meal, suddenly attacked with a *coup de sang* (those were her very words) in the left ear. Vertigo without nausea, and a condition resembling syncope ensued. She went to bed and noticed in her left ear a noise similar to the rumbling of carriages. She passed a restless night, but suffered from no special symptoms. In the following night she vomited repeatedly, and became so vertiginous that she could not sit up in bed. To her great alarm she also perceived that she could hear almost nothing with her left ear. Gradually these symptoms disappeared; two months subsequently her gait was still tottering, and she suffered from vertigo whenever she moved her head rapidly. The hardness of hearing of the left ear persisted.

A young and robust merchant, in vigorous health, while in the act of giving orders to one of his employees, fell suddenly to the ground and remained motionless. His arms and legs were in a state of perfect relaxation. When he was raised his face was pale and covered with perspiration. Nausea and vomiting ensued. Consciousness had but momentarily been obscured, and was speedily restored. The patient stated that everything whirled around him. This motion had a nauseating effect. He also complained of noises in both ears. His previously excellent hearing was considerably impaired. At a subsequent time the attack recurred several times without affecting his general health in the least.

Ménière does not regard the disease as rare, and I believe him to be right. I remember to have observed at least three such cases.

If we except the phenomena of the organ of hearing and consider the disease symptomatically, we find that it does not differ from simple vertigo. It is well known

that this affection likewise commonly occurs in paroxysms attended with intense nausea, vomiting, cold perspiration, general muscular relaxation, sudden falling, and momentary loss of consciousness. I can, therefore, not readily conceive why accurate observers should have mistaken the disease for cerebral congestion. In some cases it seems to bear a certain analogy to epilepsy, but even then we find many distinctive features, viz.: complete muscular relaxation, lack of tonic or clonic convulsions, and either persistence or but momentary abolition of consciousness. We thus perceive that in cases of violent vertigo it becomes necessary to examine the condition of the organ of hearing.

What is the anatomical basis of Ménière's disease, and how may its symptoms be accounted for?

Ménière considers it essentially a disease of the inner ear, and supposes that the seat of the affection will be found in the semicircular canals. This view is adhered to in most of the treatises on aural diseases. Let us enter more fully upon the consideration of this subject.

Many weighty reasons may be adduced to show that the phenomena are due to a disease of the organ of hearing. We find, for instance, that abnormal sensations in the ears occur simultaneously with or shortly before the paroxysms; that the latter, moreover, coincide with the beginning of an incurable hardness of hearing. We are thus led to infer the existence of a certain correlation. Ménière assumes a relation of cause and effect, from the fact that, with the exception of deafness, these paroxysms

produce no derangement of the cerebral functions. As additional points, we may mention the relation which exists between diseases of the ear and vertigo, and the possibility of producing the latter by impressions upon the organ of hearing. I consider it highly probable that in the majority of cases alterations of the inner ear will be found; but as violent attacks of vertigo may be likewise caused by affections of the middle ear—and our diagnostic means are rather imperfect—we ought to give our statements with some reserve. Moreover, we should investigate whether pathological conditions of the acoustic nerve or its roots play a part in the production of those symptoms. Ménière's view, that the disease has its seat in the semicircular canals, is based upon the result of a post-mortem examination and upon experiments instituted by Flourens. Unluckily the person of whose body Ménière made the autopsy had suffered from an essentially different disease. Of a case of true Ménière's disease no post-mortem examination has as yet been made.

A young girl, who, during her menstrual period, rode a whole winter's night on the imperial of a mail-coach, suddenly became totally deaf. On being admitted into Chomel's clinic, she suffered from continuous violent vertigo. The slightest motions produced vomiting. She died on the fifth day. At the autopsy the brain and spinal cord were found normal. The temporal bones were subjected to a very careful examination, and a reddish plastic fluid was discovered in the semicircular canals. Nothing was found in the vestibule and cochlea.*

* In Ménière's paper there are unfortunately no accurate statements as to the course of the disease. He merely refers to another paper, which I have not yet been able to find.

Is it not evident that this case differs essentially from true Ménière's disease? In the case under consideration a grave disease set in after exposure to cold, and terminated in death after five days. In Ménière's disease the paroxysms occur repeatedly, have no assignable cause, and do not affect the general health. The case is interesting, and in many respects obscure, especially as regards the cause of death. It demonstrates, at the most, that inflammation of the semicircular canals may occasion deafness and violent attacks of vertigo. But it does not appear how inflammation of the semicircular canals could occasion complete deafness in cases where the remaining portions of the labyrinth and the auditory nerve are intact. The case seems to resemble more closely otitis labyrinthica of Voltolini. No mention is made of a microscopic examination.

Moreover, it is obvious that a mere localization of the affection to the labyrinth constitutes an incomplete definition of the disease. How can we explain the sudden and paroxysmal occurrence of the phenomena? Are we to suppose that either violent congestions or repeated hemorrhages occur in the labyrinth?

We may here mention several interesting items reported by Gruber.* He says: "Up to this time I examined four syphilitic patients, who, according to their own statements, had suddenly become deaf after overheating, or simply exposing themselves to cold. They had previously either enjoyed good hearing or become but slightly hard of hearing in consequence of chronic aural catarrh. In the beginning they suffered

* Treatise on Otology, 1870, page 617.

from attacks of vertigo, which subsequently disappeared. Deafness, however, persisted and defied every method of treatment. One of the patients incidentally died of typhus fever, shortly after the occurrence of his aural disease, and an examination of his organs of hearing showed intense hyperæmia of the tympanic mucous membrane, and vascular injection and great thickening of the soft parts of the labyrinth. The fluid of the labyrinth was of a reddish tinge and copious. Are we to consider this condition as an inflammation attended with hemorrhagic exudations, and occurring chiefly in syphilitic persons?

As the subject under consideration is new and important, I beg to be allowed to mention my own observations and to call attention to the fact that besides cases analogous to those of Ménière, we find other cases constituting transition forms, in which chronic aural catarrh obviously existed as the primary affection.

The following case is analogous to Ménière's cases:—

In 1860 I was consulted by a farmer, 43 years of age, on account of hardness of hearing of both ears and humming noises of the left ear. When in his 20th year he had already noticed an impairment of his hearing. In his 23d year he had, while working in the field, so violent an attack of vertigo that he fell. At the same time he felt as if his right ear were stopped up, and he heard loud ringing noises in both ears. He vomited repeatedly. The attack disappeared gradually, but for several weeks the patient suffered from vertigo and unsteadiness of gait. Subsequently he had no similar attack. He remembered that when 12 years of age he had an attack of vertigo, which caused him to fall from a ladder. After the last attack his hearing was constantly decreasing, less rapidly at first than during the last five years. An examination showed a considerable impairment of his hearing, not only for speech and watches, but also for conduction through the bones. Both tympanic membranes were normal and movable. I diagnosticated an affection of the inner ear, and advised no treatment.

The patient had during the attack of vertigo a sensation of closure of the left ear; this symptom seems worthy of attention. Are we to assume a closure of the corresponding Eustachian tube? It is true that a sensation of fulness commonly occurs in closure of the tube. This depends upon a difference of pressure within and without the tympanic cavity. But I wish to state that in my own person a sensation of tension and fulness of the ears appears and disappears simultaneously with a clear ringing sound. I speak of the clear tone of high pitch which at times suddenly appears in the ear and dies away gradually.

The second case occurred in the person of a farmer, aged 50, of robust frame, who had never shown a tendency to disease. In 1869, on a very hot day, while engaged in the hay crop, he was attacked with violent vertigo, compelling him to lie down on the ground in order to avoid falling. He suffered from intense nausea, became deadly pale, and cold perspiration covered his face. After a few hours his condition improved, but he still had a sensation of unsteadiness and a certain tendency to vertigo, which compelled this vigorous man to abstain, especially in hot weather, from heavy work, in order to avoid an attack of vertigo.

He feared that he was threatened with apoplexy. Similar attacks recurred at times every week, at times after longer intervals. In February, 1870, the paroxysms commenced to be less marked; they caused no fall, and were not attended with nausea and cold perspiration. Immediately after the first attack the patient noticed a buzzing noise in his left ear; this subsequently never ceased completely, but became more intense before every new attack, in a manner that the patient could predict the approach of a paroxysm by the exacerbation of the noises. The hearing of the left ear became impaired by the first

attack. At present, 15 months after the accident, the patient hears still tolerably well and understands ordinary conversation.

In August, 1870, the patient consulted me on account of his hardness of hearing. Prior to that time I had but incidentally seen him, because his son had been under my treatment. Upon examination, I found both tympanic membranes dull, the right duller than the left, their lustre and concavity almost normal. By Valsalva no motion was produced in either membrane. On employing the air-douche, I could hear distant and indefinite noises on both sides, and only when the patient made a swallowing movement the air seemed to approach in short puffs. This and the examination with the bougie showed that the Eustachian tubes were not completely free.

The patient heard the ticking of a lever watch (normal hearing distance of 150 centimetres), with the right ear at 15 ctm., with the left ear at 1 ctm. By conduction through the bones, neither this watch nor a loud ticking cylinder watch was heard. Vibrating tuning fork C′ placed upon vertex, not perceived. Tuning fork C″ only heard in right ear, even when placed upon the left side of the head.

I shall now relate several cases of chronic aural catarrh attended with vertigo.

Jacob Meier, a saddler of Naenikon, aged 52 years, and in good health, consulted me on the 12th of November, 1870, on account of hardness of hearing. From his history I learned that in 1857, after exposure to cold, his right ear became painful, and commenced to discharge. The otorrhœa lasted three weeks. From that time the hearing of the right side was impaired. Two years before he consulted me he suffered from violent nasal catarrh, in consequence of which his right ear was stopped up. Whenever he blew his nose, he had a crackling sensation in his left ear, and noticed that his hearing was temporarily improved. As this procedure gave him great relief, he often resorted to it. A short time subsequently he began to suffer from slight vertigo recurring every morning. At times the symptoms were severe and attended with great nausea. He had no noises in his ears. The

symptoms of vertigo gradually subsided, but the hearing of the left ear remained impaired, and seemed to vary according to the condition of the weather. For the last year the patient has been troubled with noises in both ears. He states that up to three months ago his hearing was tolerably good. After that time, while at work in the field, he had a violent attack of vertigo. Similar attacks, attended with great nausea, vomiting, and cold perspiration on the forehead, recurred repeatedly in one week, and whenever he worked heavily. Each attack generally lasted from 1 hour to 2 hours, and then disappeared; after it the patient felt weak and miserable all day. By the first attack his hearing was considerably impaired.

Condition of the patient on the 12th of September, 1870.

Patient hears lever-watch (hearing distance 150 centimetres) in contact with auricle, with either ear. He does not hear it by conduction through the bones. Cylinder-watch (hearing distance 12 metres) at $1\frac{1}{2}$ centimetres in either ear. Not heard by conduction through the bones. Tuning-fork heard from vertex. Stopping up the ears renders the sound louder in the left ear. No increase of sound in right ear. Patient understands speech with difficulty even in close proximity. Tympanic membranes whitish and dull—in the left membrane nothing peculiar—in the centre of the right membrane a bluish atrophic spot. An examination with the catheter, air-douche and bougie shows a narrowing of the left Eustachian tube, and no, or at all events, very slight narrowing of the right tube. The auscultation sound of the left ear is distant and faint; the air does not enter; a bougie of .8 millimetres in thickness can with great difficulty only be passed up to the tympanic cavity, and is then tightly held by the tube; the right Eustachian tube, on the contrary, admits the bougie very readily.

It is obvious in this case nasal catarrh gave rise to chronic aural catarrh attended with vertigo. In making this statement we do by no means exclude the possibility of a secondary affection of the labyrinth. We noticed that in the case under consideration, the Eustachian tube of

the primarily affected worse ear had again become pervious, while the E. tube of the left ear showed a narrowing of its calibre.

The following case offers many points of interest:—

A healthy and vigorous stone-cutter, forty and odd years of age, suffered from a violent nasal catarrh which lasted about three weeks. He was advised to inhale the vapor of hot water through his nostrils. This he did twice on two succeeding evenings, every time for half an hour. The next day, October 2d, he felt well, with the exception of the nasal catarrh, and resumed his work. At nine o'clock in the morning he was attacked with violent vertigo. He had no headache, no tinnitus aurium. He continued to work till four o'clock in the afternoon. At that time his vertigo became more violent and compelled him to go home and lie down. The symptoms then abated. The next day he remained in bed. All day he heard noises of boiling water in his head, especially at its vertex, also a buzzing noise in his right ear. He felt as if his right ear were filled with water, and instinctively introduced his finger into the ear in order to remove the imaginary water. When the patient touched the region of the ears he elicited a peculiarly hollow sound. His own voice sounded uncommonly dull. The following day he resumed work, but was again attacked with vertigo of such violence that he was compelled to hold on to surrounding objects in order to avoid falling. No headache, no vomiting. The following day he remained at home and sent for me. On examining him with my watch (hearing distance 150 centimetres) I found R. 15 ctm., L. 30 ctm. The tympanic membranes showed diffuse opacities. No signs of exudation within the tympanic cavities. Not being provided with instruments, I could not at the time make a more thorough examination. I prescribed an alum gargle, and a solution of nitrate of silver, two grains to the ounce, to be snuffed up through the nostrils. The following day, October 6, the patient resumed his work. The vertigo was then abating. On October 9th, the noises in the head, the buzzing noise and sensation of closure of the

right ear had disappeared. On October 11, at half-past ten o'clock, he left his work and went into an inn, where he remained till half-past three in the afternoon, freely partaking of fermenting wine. When he reached his home he vomited. The next morning he felt well again, and left his house in order to go to his working-place; but on his way had so distinct a sensation of his ear being filled with water that he involuntarily put his finger into the ear. The sensation was attended with noises of boiling water and vertigo. While he was at work the vertigo grew very violent, compelling him to hold on to surrounding objects; his forehead was covered with cold perspiration, and vomiting ensued repeatedly. He went home and lay down; the vertigo then abated, but did not cease; the noises in his head continued, and in his right ear he heard a loud noise resembling that of crickets. He passed a quiet night. I found him out of bed the next morning. He had very little vertigo, but the noises of boiling water still continued; the sensation of his ear being stopped up by the presence of water had ceased. He heard my watch R. & L. at the distance of three centimetres, but could not hear it by conduction through the bones. The tuning-fork C′ was equally and but faintly heard on either side. Closing the ears produced no alteration in the intensity of the sound. His hearing for speech was not materially disturbed, yet he stated that he formerly had heard better. Upon examination with the catheter I found both Eustachian tubes narrowed. I was in doubt as to whether air could be forced into either ear. A bougie of .9 millimetres in thickness could be passed up only with difficulty to the cavity of tympanum, and was not easily removed. The symptoms of vertigo were entirely gone after a few days; his general health was good, and at the end of the week, October 18th, he resumed his work without being compelled to stop it again. On October 16 he heard my watch R. 3, L. 6 centimetres. A cylinder watch of 12 metres hearing-distance R. 68 centimetres, L. 58 centimetres. By conduction through bones on both sides, November 7, lever watch, R. 25, L. 2 centimetres; cylinder watch, R. 80, L 48 c. The lever watch, when placed upon the right mastoid

process, was very faintly heard. Both cylinder watch and tuning-fork were heard by conduction through the bones, but the perception of sound became less distinct when the ears were closed. The examination with the catheter yielded the same result as before, except the bougie passed into the right Eustachian tube more readily than into the left. In the left ear the handle of the hammer was somewhat injected, otherwise I found nothing peculiar in either ear. Now the left ear was the worse, formerly the right. According to the positive statement of the patient, his hearing for speech had become worse since the last examination, though his hearing for watches had improved. By objective examination I was unable to confirm these statements. Vertigo had entirely disappeared, there was not the least unsteadiness in his movements, but the noise of boiling water still continued throughout the head, especially at the vertex. Prior to this affection the patient never had suffered from noises in his ears, and according to the testimony of his friends we may assume that formerly he had heard better. The patient was unwilling to submit to special treatment, for want of time. I may add that at present, *i. e.*, one year later, his condition is the same. The noise of boiling water still continues, and his hearing is somewhat, but not materially, diminished.

This case is certainly very instructive as regards the connection of chronic aural catarrh with vertigo. The uncommonly violent and continuous nasal catarrh, which the patient disregared despite the inclement season of the year, had obviously extended through the Eustachian tube to the middle ear. At first the patient complained of continuous vertigo, subsequently of noises in the ears and head (these noises afterwards did not leave the patient), and of a sensation of the ear being filled with water. He furthermore stated that his own voice

sounded dull, and that on touching the aural region he heard a peculiar hollow sound. This furnishes a proof of obstruction of the Eustachian tubes and of resonance in the cavity of the tympanum. It is noteworthy that the renewed violent attack of vertigo which occurred on the 12th of October was preceded, exactly as the cases cited above, by a sensation of obstruction in the ear, attended with noises of boiling water. From the connection of the phenomena, it cannot be doubted that the vertigo proceeded from the ear. But the question of the more intimate relation of vertigo to chronic aural catarrh, so important to the aurist in a diagnostic point of view, cannot yet be considered solved.

Are we to seek the cause in variations of intra-auricular tension affecting the labyrinth? Variations of tension ensue by increased pressure upon the drumheads, after closure of Eustachian tubes, also by pressure of mucus upon the fenestræ of the labyrinth. Or are we to assume that vertigo is produced by congestive or perhaps inflammatory conditions of the labyrinth? We may adduce reasons for either possibility: for the first, the sensation of obstruction which frequently accompanies the attack of vertigo; for the second, the subjective noises. But we are unable to decide which of the two causes plays the more important part in the production of vertigo. At all events, it is important, as regards treatment and prognosis, not to content one's self with the convenient supposition of increased intra-auricular pressure, and not to lose sight of the other possibility.

I may still mention the two following cases of affections of the middle ear attended with vertigo.

Under the title, "Apparent Cerebral Symptoms occurring in the course of purulent inflammation of the tympanic cavity, and their cessation after one catheterization: a contribution to the clinical significance of increased intra-auricular pressure," Moos* reports a case of otitis media purulenta, with painfulness of the mastoid process, and perforation of the membrana tympani on the 12th day of the disease. On the 14th day the patient began to suffer from vertigo, vomiting, and headache. These symptoms, which on the 26th day were followed by a slight contraction of the pupil of the same side, continued to the 35th day, when Moos was consulted and applied the catheter. The patient felt relieved, and was in the following night for the first time free from vertigo and vomiting. From that time these symptoms did not return, and the condition of the patient improved very rapidly. The perforation of the membrana tympani was, at all events, small (Moos did not succeed in finding it), and the now and then interrupted discharge very slight. Moos is inclined to account for the vertigo by an increase of intra-auricular pressure, and to refer the contraction of the pupil of the otic ganglion (?). The supposition that retention of inflammatory products may have caused, in the vicinity of the tympanic cavity, a hyperæmia of the brain and its meninges, seems to him

* Archiv f. Ohrenheilkunde, II. p. 197.

far less probable, especially on account of the rapid cessasation of the symptoms.

In Gruber's Treatise on Aural Diseases I find the following passage:—

In rare cases violent vertigo occurs in persons with defective drumheads. Such patients generally plug up their auditory canal instinctively. I saw a patient, who was in the habit of using a cotton plug, become very unsteady in standing and walking whenever he had forgotten to stop up his ear. Without the plug he did not dare to walk in the street, as he was obliged to hold on to objects in order to avoid falling. It seems that these symptoms arise from a preponderance of the outer atmospherical pressure, the Eustachian tube proving insufficient for the ventilation of the tympanum.

I consider this explanation of Gruber incorrect and substitute the following for it.

If the patient was in the habit of having a plug in his ear, he might have become accustomed to the pressure made upon the ossicles and the remnants of the membrana tympani; and vertigo might have ensued whenever the pressure was suddenly removed.

We shall now consider the diseases of the labyrinth. Voltolini* has the merit of having first indicated a group of symptoms occurring chiefly in children and constituting one of the most frequent causes of deaf-muteism. He defined this affection as acute inflammation of the membranous labyrinth.

* Monatsschrift f. Ohrenheilkunde, 1867, page 9; 1868, page 91.

Up to this time no case of this affection has come under my notice, but I observed that of the deaf-mute children whom I had the opportunity of examining the majority had a tottering gait. This I considered as a proof of the intimate, though yet unknown connection of vertigo with the organ of hearing, for in these children the mental faculties were intact, and there existed no other impairment of innervation, either motory, sensitive, or sensuous.

Voltolini described such cases more fully, not only from their history, which, considering the age of the patients, must be very imperfect, but also from actual observation of several cases. Dr. Reichel,* physician to the Children's Hospital in Breslau, also described a case.

Commonly the disease sets in suddenly; the children suffer from fever, have a hot and burning head, and become uneasy and excited. Sometimes vomiting ensues. Consciousness generally vanishes either totally or partially in the first twenty-four hours; at the same time the patients are very much excited and delirious, toss about in their beds, and scream violently. After two to four days they become comatose. This condition lasts about two to four days, whereupon consciousness reappears. The children recover their senses very rapidly, but when they first attempt to walk they are found to stagger. While the tottering gait gradually disappears, incurable deafness develops with more or less rapidity.

* Berlin. Klin. Woch., 1870. No. 24.

It cannot be doubted that this group of symptoms, which we must consider a disease sui generis, really exists, though its pathological basis is not yet known. As the disease generally terminates in recovery, we have no opportunity for anatomical research. But the impulse is given, and the solution of the problem may be expected. Voltolini is of opinion that the seat of the affection may be ascertained by exclusion and without an autopsy.

He says, an affection which after its subsidence leaves behind deafness and a tottering gait, but no other nervous disturbances, and which during its course was not attended with symptoms indicating a diseased state of other parts of the nervous system, cannot be classified under the head of the ordinary forms of inflammation of the brain or its meninges. No serious doubt can be raised against the assertion that the disease constitutes an entity, and depends upon pathological conditions of the organ of hearing. Taking this for granted, and considering that diseases of the middle ear never cause such complete deafness, we conceive that the seat of the disease must be found either in the labyrinth or the auditory nerve. The latter possibility Voltolini rejects for various reasons. If, however, we regard the labyrinth as the seat of the disease, we cannot readily explain the fact that the affection occurs constantly on both sides. For this reason I am led to mention the possibility of a central cause, *e. g.*, an affection of the ventricles of the brain (in particular of the fourth ventricle), for which there seems to exist a certain predisposition at an early age.

I base this supposition upon the interesting case* reported by Professor H. Meyer, which shows that in thickening of the endyma resulting from disease of all the ventricles, the striæ acusticæ, on account of their superficial situation, may become permanently altered. (The ganglion of the acoustic has also a very superficial situation.) At all events, I do not agree with Voltolini, who considers the question as to the seat of the affection as solved.

I shall here remark, that our knowledge concerning the origin of the acoustic is not sufficiently established to allow us to reach any conclusions as to its pathology.

What we now know of the acoustic is substantially this:†

The acoustic nerve as one undivided cord passes at the inferior boundary of the pons into the medulla oblongata. It then divides:

1. Into fibres to the striæ acusticæ. The latter take their course around the restiform body along the floor of the fourth ventricle to the median line, where they disappear. Their further course is not known.

* Virch. Arch. xiv., page 551. In the autopsy on the body of an intelligent deaf-mute, Meyer found both organs of hearing and the acoustic nerves completly normal; the endyma of the ventricles of the cerebrum and cerebellum thickened; the floor of the fourth ventricle completely leveled by the thickened endyma—of the striæ auditivæ no trace. As the cause of this condition Meyer considers meningitis interna intra-uterine. It is, however, possible that the disease occurred after birth. We know how difficult it is to determine whether a child was born deaf, or lost its hearing in the first year of its life. In a second case Meyer found that, in consequence of thickening of the endyma, the fourth ventricle was shallow, and the striæ auditivæ considerably reduced in size.

† For this résumé I am indebted to Dr. Huguenin, of Zurich.

2. Into fibres to the ganglion of the acoustic.

3. Into fibres running to the funiculus cuneatus et gracilis (sensory).

4. Into fibres to the restiform body (motory).

5. Into fibres to the accessory acoustic ganglion of Stilling. This is a small ganglion situated beside the restiform body in the cerebellum.

6. The acoustic ganglion is connected by means of the so-called acoustic cord with the cortex of the insula Reilii (centre of speech) near the fossa Sylvii. The acoustic cord constitutes a bundle of fibres which can be traced posteriorly to the acoustic ganglion in the medulla oblongata, where it becomes lost.

Now, if we seek for an explanation of auditory vertigo, we must above all take into account the experiments and hypotheses on the significance of the semicircular canals.

In 1817, Flourens* published his well-known experiments. He observed that in birds and rabbits, after cutting the semicircular canals, a series of peculiar movements, so-called compulsory movements, ensued. The cutting of the external semicircular canal was followed by movements of the head from right to left; of the superior canal by movements from above downwards with a tendency to fall forward, and of the posterior canal by movements from below upwards with a tendency to fall backwards. If the animal is left to itself these movements after a while cease, but if irritated in any manner

* Recherches expérim., 2me éd., 1842, p. 442.

they will reappear, and that, too, the more violently the more the animal is disturbed. Upon cutting several semicircular canals a combination of the above detailed movements will ensue. Flourens says he has found that the ampullary nerves spring with three separate roots from the pons, the cerebral and the cerebellar peduncles, or the restiform bodies, and that after a section of these parts of the brain exactly the same movements will follow which result from cutting the corresponding semicircular canals.

These statements, however, have not been corroborated by anatomical research. The described movements are said not to ensue by the mere opening of the bony canals, but only after cutting or pricking the membranous canals. Upon destroying the cochlea and the structures of the vestibule, Flourens produced deafness, but no anomalies of motion; while by opening and cutting the semicircular canals he could not induce deafness!*

Harless† says that his experiments with doves substantially corroborated the loss of co-ordination after removal of the one or the other semicircular canal, but that the form of the disturbance differed from that observed by Flourens.

Czermak, in his experiments with doves, agrees with Flourens.

* How could the cochlea and vestibule be destroyed without impairing the functions (whatever these may be) of the semicircular canals? And yet Goltz cites these experiments in support of his hypothesis.

† Handwörterbuch der Physiologie, IV. p. 422.

From the fact that the acoustic nerve is distorted by the operation, Brown-Séquard endeavors to explain the observations of Flourens. Séquard found that, as mechanical irritation of the processus cerebelli ad pontem, so also irritation of the acoustic nerve produced rolling movements. With the aid of clinical observations he reaches the conclusion that the acoustic nerve, by reflex action, may give rise to convulsions, vertigo, and other symptoms of derangement of the cerebral functions.

Schiff* says: "In the experiments with rabbits, the cutting of the acoustic nerve gave no indication of pain as long as the fifth pair was uninjured. The movements of the animals were entirely normal while the lateral parts of the pons remained intact. Frogs also show normal motility after the cutting of the nerve without torsion." In view of these facts he deems it surprising that Brown-Séquard should assert that wounding the acoustic in mammals produces whirling and rolling of the body and convulsive movements which last as long as the ani mal survives. He says, moreover, there is no foundation in the hypothesis that the acoustic nerve consists of two nerves, the one supplying the organ of hearing, the other regulating the peculiar movements which Flourens obtained after cutting the semicircular canals.

The case of Signol and Vulpian,† so frequently cited, is of no value, I think, in reference to the semicircular canals; at the most it can be brought forward as proving

* Lehrbuch der Physiologie, p. 399.

† Gazette Médicale de Paris, 1861, p. 716.

that an injury of the labyrinth may produce disturbances of motory co-ordination.

But also against such a conclusion weighty objections may be raised. For even if the brain were found intact by microscopical examination, its physiological function might have suffered nevertheless; and this would not be surprising, on account of the destruction of the bones of the skull and the formation of sequestra.

As the experiments of Goltz* offer many points of great interest to the aurist, and are of fundamental importance with reference to the subject under discussion, I shall consider them somewhat in detail.

Goltz found that frogs, in which both acoustic nerves had been cut, frequently wallowed in their attempt to move, and could with difficulty only preserve their balance. He obtained the same result after destruction of the labyrinth, which constitutes the extra-cranial, peripheral expansion of the acoustic nerve. Such an effect is certainly very striking if we are to consider this nerve exclusively as the nerve of hearing. Moreover, Goltz's experiments do not confirm Schiff's observations. Goltz, in repeating the experiments of Flourens, did not only cut the semicircular canals, but also chiseled out portions of them, supposing that, in accordance with the observations of Flourens, the phenomena would become much more pronounced than after mere cutting. The animals operated upon showed permanently all the symptoms described by Flourens. Generally their heads were awry

* Pflueger's Archiv. f. Physiol., III. p. 172.

and moved to and fro. On endeavoring to run forward, they would constantly execute the so-called "mouvement de manège," move their heads very forcibly, and, violently flapping their wings, sometimes roll backwards, as if attacked with vertigo. Their attempt to pick a pea was generally unsuccessful. But while the animals had great, often insurmountable difficulty in seizing outside objects, for instance, peas, they would easily hit all the accessible parts of their own body and plume themselves perfectly. Goltz here remarks that the simple reflex mechanism as called forth by the sensation of twitching, remains undisturbed, while the more complicated reflex action, induced by the sight of food, *e. g.* a pea, becomes materially impaired.

Goltz, moreover, made the observation that slightly supporting the head proves sufficient to impede its movements. If we hold up straight the head of a pigeon and dip it into a bowl of water in a manner that the beak but slightly touches the bottom, the animal, if thirsty, will drink for a certain time, without changing the position of the head. This also occurs when the body of the pigeon is fixed and its beak thrust into a dish of peas. Then the head of the animal will not immediately turn to one side, though it is not held. We also notice that the pigeon repeatedly draws its head entirely out of the mass of the peas, without changing the correct position, and then thrusts it back again, though not with as much precision as formerly.

Goltz lays particular stress upon these observations,

and is of opinion that the movements of the animals depend upon the disturbance of the equilibrium and upon the vertigo resulting from it. He also says that, if a person is suffering from vertigo, the least support will suffice to enable him to regain his balance, whereupon the tottering movements immediately cease.

If we assume, Goltz continues, that the essence of the phenomena under consideration depends upon the loss of equilibrium, let us then consider under what circumstances this equilibrium may be disturbed: 1. This may occur whenever the centripetal impressions by means of which the central organs regulate their activity are either suspended or impeded. 2. Whenever the central organ is impaired. 3. Whenever the centrifugal apparatus is impaired.

Goltz is of opinion that in his experiments he could always exclude the conditions 2 and 3. He says it is obvious that by the operation neither a muscle nor a nerve supplying a muscle is injured. The existence of a permanent change in the structure of the brain cannot be assumed. Transitory irritation or reflex action will not account for the persisting motory disturbances.

This, it seems to me, is the weak point in the argument of Goltz, since from even a most careful microscopic inspection no conclusions can be drawn as to the functional integrity of such subtle parts. But Goltz continues as follows:—"We excluded two possibilities, and one possibility remains. We cannot, however, suppose that an anomaly of hearing causes these striking phenomena, the duration of which does not allow us to refer them to

reflex irritation of the acoustic nerve. We must therefore infer that the semicircular canals have the special function of the preservation of the equilibrium." According to Goltz they constitute a kind of central organ for the balance of the head, and thus indirectly of the whole body, and play a more important part in regulating the carriage and movements of the head than both the muscular sense and the eye. (?)

As to the manner of their function Goltz theorizes as follows:—He supposes that the nervous terminal apparatus of the ampullæ responds to excitation by pressure or tension, thus resembling the special cutaneous nerves for the transmission of impressions of weight. The fluid of the semicircular canals will, according to well-known physical laws, most forcibly distend the inferior portions of the walls, thus causing a definite form of nervous excitation for every position of the head.

Goltz does not discuss whether the semicircular canals perform at the same time functions of audition. Perhaps it seemed strange to him that contiguous portions of the labyrinth should so widely differ in their functions. Perhaps he also found that the terminal nervous apparatus of the ampullæ, so far as yet known, can be considered as an acoustic apparatus rather than an apparatus of equilibrium. Physiologists may decide* whether

*Malinin (Centralbl. f. med. Wiss., 1866, No. 43) supposes that the semicircular canals play a part analogous to that of the black pigment of the eye, by producing a complete interference of the sonorous waves, which pass simultaneously into both openings of each semicircular canal, and meet in its middle.

in the structure of the semicircular canals a basis for the new function can be found, and here it should be remembered that the membranous canals are applied to the convex side of the bony canals. Furthermore, it should be taken into consideration whether these two functions will not interfere with each other, *i. e.*, whether we can consistently locate a kind of manometer in an organ whose contents are continually subjected to variations of tension by sonorous waves. From the experiments made by Schmidekam with the sirene, it appears that an intense sound can, by itself, occasion violent vertigo, attended with vomiting.

The correctness of the hypothesis of Goltz may, perhaps, more thoroughly be examined from a pathological point of view than by physiological experiments; and I wish to lay particular stress upon the following three points:—

A. If the acoustic nerve not only performs functions of hearing, but also regulates the equilibrium, there should, in case of atrophy of the nerve, not only deafness ensue as a consequence, but also the above-mentioned impairment of equilibrium, particularly if the atrophy occurs on both sides; but if only one side be injured, then, according to Goltz, loss of equilibrium would be as unlikely to follow as blindness from the loss of one eye. Until now this correlation has not been noticed; in literature we find many cases of atrophy and amyloid degeneration, especially of the ampullary nerves; but little or nothing is stated in regard to the symptoms during

life, especially with reference to disorders of equilibrium. As atrophy of the acoustic nerve occurs frequently, we shall have the opportunity of testing the correctness of Goltz's hypothesis.

B. The above applies also to cases of absence of the one or the other semicircular canals, and—

C. To cases in which both the membranous labyrinth and the semicircular canals are completely disorganized.

We find pertinent observations in the results of the autopsies made by Voltolini, to whom we are indebted for most of our knowledge of the pathological anatomy of the labyrinth. I do not find mentioned notable disturbances of the equilibrium, vertigo, tottering gait; but to these points no special attention seems to have been paid.

However convenient and enticing Goltz's hypothesis may be for the explanation of the vertigo under consideration, I must, nevertheless, regard it as hazardous, and resting upon an insufficient basis; and the pathological facts are not as much in favor of Goltz's views as he supposes. If, for instance, he asserts that there exists a series of facts demonstrating that persons may suffer vertigo, vomiting, and derangement of equilibrium whenever there is increased pressure of the fluid in the semicircular canals, or whenever the latter are injured, or in order to confirm it, refers to Ménière's disease and the so-called symptoms of nervous irritation in aural catarrh, then I must refer to the foregoing evidence that this correlation is merely hypothetical.

Goltz cites a case of perforation of the membrana tympani in which sudden vertigo followed careless syringing, and says: "Such persons state positively that the attack of vertigo is not attended with any sensation of sound." This important assertion, upon which the following deduction is based, I cannot accept without further proof. I must, as opposed to this, state that tinnitus aurium belongs to the regular complex of symptoms which constitutes vertigo, and pronounced cases of vertigo are not only attended with, but also ushered in by noises in the ears. I can believe that in cases of slight vertigo the patients whose attention has not been called to the noises may not notice them. But hereafter I shall examine this point with particular care.

Thus, also, the further reasoning of Goltz that the nerve, which may cause vertigo, when irritated by syringing, cannot be the acoustic, because its excitation would give rise to sensations of sound, and that there must be in the inner ear the expansion of another nerve of peculiar function, becomes void.

Setting aside the special significance of the semicircular canals in the preservation of equilibrum, and considering the acoustic exclusively as the auditory nerve, we shall certainly be no more able to give a sufficient explanat io of the auditory vertigo than to account for many cases of gastric vertigo. There is still wanting the link of connection between the acoustic and the central organ. And we may also question, in the cases under consideration, whether vertigo always depended upon excitation

of the acoustic nerve. If, for instance, a column of cold water in the external auditory canal causes violent vertigo, while a column of warm water of the same height has no such result, we must regard the nerves of the external auditory canal and membrana typani (vagus and trigeminus) as the media by which the vertigo is effected, unless we imagine that the sudden impression of cold produces a considerable hyperæmia of the middle ear.* In the great majority of cases, however, where, to all appearances, increased pressure or congestive conditions of the labyrinth exist, it seems most natural to regard the acoustic as the medium by which the vertigo is effected. In what way may the auditory nerve be connected with the mechanism of equilibrium? Can it be supposed that hearing is of great significance in the appreciation of the position of the body? I do not believe it, and cannot consider auditory vertigo as being essentially sensuous, analogous to visual vertigo; for auditory vertigo does not seem to originate in delusions, but in a direct reflex from the acoustic nerve upon the centres of co-ordination and of equilibrium.

There is no satisfactory explanation of this mutual relation. We might ask whether this relation constitutes a supposed and necessary connection, or whether it is based upon an incidental contiguity of the nerves, in other words, whether vertigo appears only in certain

* Bonnafont cites a case in which sudden incurable deafness ensued in consequence of exposure of the head to great cold. (Traité des Mal. de l'Oreille. Paris, 1860, p. 365.)

states of particularly severe excitation of the acoustic nerve. It is perhaps of significance that the acusticus originates in the medulla oblongata, which is considered as the particular seat of the more complicated movements, and probably also contains the centre of equilibrium. We have, moreover, seen that fibres of the acoustic nerve pass into the corpus restiforme, but we do not know for which purpose. Physiologically, there seems to exist a certain sympathy between hearing and motion. As a proof of this we may call to mind the sudden start and turning of the head on hearing a loud report, or also the effect of rhythmical music. (?) Unfortunately we know but *little* of the mechanism of reflex actions, and nothing of the part played by hyperæsthesia of acoustic nerve which so frequently occurs in ear patients.

In this respect there is much of interest in the above-mentioned case of hemiplegia in consequence of the air-douche. The same cause which usually produces vertigo, gave rise in this case to an affection of the motory nerves.

To-day I made again an interesting observation of this kind.

A young lady suffering from catarrh of the middle ear and stenosis of the Eustachian tubes, was frequently attacked with vertigo when injections were made through the tubes according to Gruber's method. Only a minimum quantity of the liquid (zinc. sulph. gr. iss. ad aq. ℥ j.) reached the tympanum on account of the stenosis; and it was evidently the tension of the column of air

forced into the cavity of the tympanum in front of the liquid, which caused the vertigo. To-day when I made Gruber's injection on the right side, the patient became for a moment vertiginous and had a tendency to fall to the right side. She remarked particularly that she felt drawn in that direction, and when she rose I noticed this tendency. She did not have the sensation of her right side being weakened; but in analogy with the case just mentioned we are led to suppose, that starting from the ear there was a momentary impairment of the motory innervation of that side, and a consequent disturbance of the equilibrium, which caused vertigo and a corresponding involuntary motory innervation.

At all events, such observations possess great interest, inasmuch as they tend to show that there exists a constant relation between the affected ear and the side toward which the body inclines.

Zürich, October, 1870.

FOUR CASES OF GUNSHOT WOUNDS OF THE EAR.

By S. MOOS.

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Translated by J. H. Pooley, M.D., of Yonkers, N. Y., and Thomas R. Pooley, M.D., of New York.

Case I.—*Left-sided laceration of the integumental and cartilaginous ear—Partial splintering of the bony parts—Suppurative inflammation of the external and middle ear—Perforation of the membrana tympani—Adhesion of the same to the promontory—Peripheral necrosis of the mastoid process—Abscess burrowing under the sterno-cleido-mastoid muscle—Effusion of blood in the lining membrane of the inner ear—Hemorrhagic infiltration of the perineurium of the nerves between the lamina spiralis ossea—Absence of vertigo and of subjective sensations of hearing during life—Total deafness on the left side.*

B. E., a Prussian soldier, received a twofold wound at Wörth, Aug. 6th. One shot went through the right knee, the other implicated the ear. On the 17th of August, eleven days after the injury, he was brought to the hospital under my charge. There was already suppuration in the knee-joint, which later required the amputation of the lower third of the thigh; a short time afterwards, on the 6th of September, death followed.

At his reception there were visible in the left ear the following phenomena: the bullet had glanced over the soft parts in front of the tragus and the tragus itself, and had produced a shallow, granulating, pretty large loss of substance before the left ear; it had torn away the skinny and cartilaginous part of the external auditory passage in such

a way that the posterior wall of the osseous canal was struck where the same is joined to the mastoid process. The coverings of the mastoid process itself were pale, swollen, very slightly tender on pressure. The swelling extended about half an inch below the mastoid process, under the soft parts which covered it; upon pressure with the finger, and drawing the finger from above downwards towards the mastoid process, pus issued at the spot where the ball struck, and escaped into the external meatus. Later a few small splinters of bone appeared at the same spot. After their removal an irregular opening was perceptible, through which the probe passed somewhat downwards and backwards into a rough cavity, which no doubt belonged to the lower part of the mastoid process. At this spot the ball had probably struck and rebounded, for the lower portion of the auricle, lobulus, etc., were uninjured, nor was any wound of exit visible, and the ball was nowhere to be found. The integumentary layer of the external meatus was, on the admission of the patient, so much swollen that I could not obtain distinct results with regard to the condition of the membrana tympani. I could only perceive that it was perforated, because the patient was able, by the Valsalvian experiment, to force pus from the cavity of the tympanum into the external meatus. Only after the lapse of some time, and disappearance of the swelling, was a small perforation visible in the anterior upper part. The rest of the membrane was thickened, grayish red, the hammer not visible. The patient had lost the sense of hearing completely on this side: loud conversation on this side not being understood, and the bone conduction being absent, both with the watch and tuning-fork. The patient never complained of persistent subjective sensations of hearing, nor ear-ache. I must, however, leave it undecided whether these symptoms, if they really existed, were not overpowered by the excruciating pain in the knee, which, in spite of all treatment, persisted day and night with unabating violence. The described symptoms remained much the same until death supervened; only the exudation grew less, and the loss of substance in the region of the tragus became smaller.

The *examination of the petrous bone* showed in front of the left ear a roundish flattened wound as large as a five-cent piece; much pus in the external meatus; the skinny and cartilaginous parts mostly torn away. Small bone splinters on the posterior wall. After their removal an irregular opening appeared, which communicated, inferiorly and posteriorly, with a cavity the size of a hazelnut. The walls of the sinus rough, and formed merely by the carious substance of the mastoid process. Downwards the cavity communicates with a fistulous passage, which can be followed for a short distance below the sterno-cleido-mastoid muscle. The integumentary covering of the auditory canal is thickened, likewise the membrana tympani, which is of a grayish-red color, and no part of the hammer is perceptible; but anteriorly and superiorly an opening as large as a lentil is seen, through which a probe can be passed into the bony tube. The latter is unchanged; but the canal contains pus. The rest of the membrane is to a great extent attached to the wall of the labyrinth. The mucous membrane covering the ossicles as well as that in the antrum mastoideum is pale, but much thickened. The antrum contains much pus. No injection anywhere. The foot-plate of the stirrup (laid bare from the vestibule) is movable. The periosteum of the vestibule and the cochlea is hyperæmic; further, the membranous semicircular canals and the ampullæ have a rust-colored appearance. The anterior osseous and membranous semicircular canals are normal.

The microscopical examination of the inner ear, which I made with the kind assistance of Prof. Julius Arnold, showed the periosteal covering of the external wall of the cochlea interspersed with numerous larger and smaller extravasations of blood, of which the larger ones appear the size of a pea; in the tissue are found numerous larger and smaller globular formations, of a brownish-red color, and red blood-corpuscles, single and in groups, the same are imbedded in meshes in the reticular connective tissue of the periosteum. Most striking is the change in the vessels, which are surrounded by a broad sheath, containing numerous red blood-corpuscles. In the membrana basilaris the large brownish-red bodies are absent; but there are here numerous groups of red blood-corpuscles. They are especially numerous under the teeth of the first row, and on the inner side of the entrance of the nerves. At this spot there are isolated brown-red cells, which are found again in the periosteal covering of the verticular surface of the osseous lamina spiralis. The same conditions appear on the membranous parts of the ampullæ, and of those semicircular canals which already macroscopically present a rust-colored appearance. The membrana propria of the parts mentioned is likewise interspersed with globular, brownish-red bodies and groups of red blood-corpuscles. The fascicles of connective tissue stretched between the periosteal covering and the membrana tympani of these parts showed similar conditions of hemorrhage and pigmentation. The external coverings of the blood-vessels

are filled with red blood-corpuscles. The membrana basilaris of the homogeneous coat of the membranous semicircular canals (especially of the anterior), is thickly beset with transparent globules, which Lucae * has, erroneously, it seems, described as being pathological, and attempted to explain their clinical significance, but I may mention that they are found in the semicircular canals of persons of normal hearing. The epithelial covering of the semicircular canals shows nothing special, while the segment of the auditory nerve lying in the modiolus shows a pure white color, and a normal condition of the perineurium externum and internum; the bundles of cellular tissue which cover the nerve fibres, extending between the plates of the lamina spiralis ossea, are thickly beset with blood-globules and pigment corpuscles.

In many places the extravasations are nearly as large as a pin's head, and separate the nerve-fibres from one another; in consequence of the changes described in the perineurium the nerve-fibres appear as red and brown strings lying between the plates of the lamina spiralis ossea. I will further mention that on several of the brownish-red pigment globules an outline could be perceived which contained several small round bodies. The same have the shape and size of the red blood-corpuscles, and the whole makes the impression as if the layer of globular formations either were to be considered

* Concerning Certain Formations in the Semicirclular Canals of the Human Ear.— *Virch. Arch.*, vol. xxxii.

as conglomerates of red blood-corpuscles, or as larger cellular formations containing greater quantities of red blood-corpuscles. They correspond entirely to those cells containing blood-corpuscles which are found in the spleen and other organs. In conclusion I may mention that that part of the sulcus lateralis covering the mastoid cells and its immediate neighborhood was strongly hyperæmic, in some places even this region showed circumscribed black-blue and black-brown spots, from the size of a lentil to a pea. Upon closer examination it was found that the hyperæmia depended, to a considerable extent, on congestion of the diploic substance. Whether the more circumscribed blackish-brown spots represent suggillations in the bone, which must be brought in causative relation to the concussion of the bone resulting from the gunshot, I do not undertake to decide. *In the present case we have disease of all parts of the organs of hearing with the exception of the tubes.* The suppurative inflammation in the external meatus, the more peripheric caries, and necrosis of the mastoid process, the perforation of the membrana tympani, the thickening of its outer layers as well as of the mucous membrane of the cavity of the tympanum and the cells of the mastoid process, the adhesion of the membrane to the walls of the labyrinth, all these conditions present nothing striking in consideration of the wound. Most remarkable appears the effusion of blood into the membranous structures of the internal ear, and the hemorrhagic infiltration of the perineurium of the nerves lying

between the lamina spiralis ossea, with the exception of the anterior semicircular canal, that is, just the canal which lies next to the cavitas tympani. As the tympanum was free from all hyperæmia and hemorrhage, it is clear that we have had a hemorrhage in the labyrinth, the consequence of concussion of the petrous bone by gunshot. This represents a change analogous to the hemorrhage in consequence of isolated ruptures of the choroid from blows on the eye. The absence of bone conductions for the watch and tuning-fork is explained by the conditions described. Especially interesting for the diagnosis of labyrinthine diseases are the results of the experiments with the tuning-fork. Although in the described changes of the external and middle ear, all the required conditions for resonance on the left side were present, the patient nevertheless heard the tuning fork C″ vibrating on the frontal bone only on the right side. The constant absence of all subjective sensations of hearing remains very strange; probably they were nevertheless present, but were masked on account of the persistent violent pain in the knee. More likely a complete paralysis of the nerve may have existed, and thus the absence of subjective sensations of hearing been caused. In proof of this assumption it is only necessary to take into account the microscopical conditions. The cellular connective fibres lying between the plates of the lamina spiralis ossea were thickly strewn with blood-corpuscles and pigment globules, even in many places the extravasations were the size of a pin's head, and crowded the nerve-

fibres asunder. Not less surprising is the fact that no dizziness was present. We might certainly object that the patient, on account of the wound in his knee, was obliged to lie continually on his back, but then the dizziness would have appeared when he was raised up. Thus, for instance, a patient at present under my care, who suddenly lost his hearing for musical tones on the left side, and on account of dizziness and vomiting had to remain in bed for weeks, had vertigo as often as he moved his head.

The case above described forms a striking contrast to the observations published by Ménière. The pathological conditions were somewhat similar to those found by Ménière in the autopsy of one of his cases, free exudadation, but no hemorrhage visible to the naked eye, being present; but the extension of the affection of the inner ear was certainly not less considerable, with the exception of the anterior semicircular canals. If the semicircular canals, as Goltz thinks, in a normal condition represent, as it were, to a certain extent the sense of equilibrium in regard to the movements of our body, would it then be possible for vertigo to be absent in changes such as we have noticed in this case?

Case II.— *Gunshot wound of the left ear from behind forwards— Injury of the mastoid process, of the bony and integumental meatus, of the tragus, and a large extent of the corresponding side of the face— Cure by cicatrization and consequent obliteration of the external meatus—Remaining paralysis of the left side, and complete deafness on the left ear—Probable disease of the labyrinth.*

This case forms a vivid illustration, and at the same time a useful, practical addition to the former one.

Mr. G., Volunteer of the Baden Army, at the siege of Strasbourg, September 2d, 1870, received, from the ball of a Chassepot, the injury mentioned in the heading. In consequence of the violent concussion he fell immediately to the ground, lost consciousness, which gradually and completely returned in the next twenty-four hours. The following six days he suffered from violent pains in the forehead and occiput. Since then the pain has ceased. He suffered in the beginning from dizziness on rising from the bed, and also at first during his convalescence; with returning strength this disappeared, and since then he has been free from any brain symptoms. I saw him on the 27th of December, when the wound had already completely cicatrized. He wished me to say whether it was possible to open the closed up meatus by an operation, and restore the lost power of hearing.

Results of examination. 1st. A cicatrix the size of a three-cent piece, flat, of a reddish color (site of wound of entrance), on the lower half of the mastoid process. 2d. A similar but smaller one inferiorly and posteriorly to the lobule, in the indentation between the angle of the lower jaw and the mastoid process, resulting, according to the patient's statement, from a previous abscess. 3d. A large, more radiating cicatrix on the left half of the face, commencing just in front of the external meatus, a smaller offset of it running toward the temple; a larger one spreading toward the left ala of the nose. The tragus was gone, the antitragus and the lobule were drawn by two small bands of the cicatrix toward the external meatus. The meatus was funnel-shaped and narrow, so that a blunt-pointed probe could not be pushed beyond the bottom of the funnel. For the same reason examination with the mirror was impracticable. As the ball had injured the soft parts of the face in the direction of the facial nerve, there was a corresponding paralysis of the muscles. The perception of the direction of sound was completely lost, and at first was very distressing to the patient, as he constantly thought every sound came from the right side; by degrees

he learned to correct this. Tuning-forks of different pitch, vibrating upon different points of the bones of the skull, were, in spite of repeated experiments, perceived only on the sound side. When a watch of 30′ hearing-distance was pressed on the temple and the wounded ear, the patient could not be certain whether he perceived the ticking or not, only after repeated experiments he stated that he heard the watch on the temple, but much weaker than on the right side. The watch taken between the teeth was only heard on the sound side. Words spoken moderately loud, through an ear trumpet, were not understood when the right ear was closed. The Valsalvian experiment could be carried out only imperfectly on account of the facial paralysis. The introduction of the catheter was easy; the simultaneous ausculation could, however, on account of the obliteration of the external meatus, not be done with the usual success. Patient felt nothing from blowing air into the middle ear, nor had this operation any influence on the acuteness of hearing.

In the beginning, *i. e.* in the first six days, he had no subjective sensations of hearing, then for several weeks now and then knockings in the depth of the ear, and not till later, about a fortnight ago, a slight hissing noise, like the boiling of water, but never persistent, at present only now and then during the night, often for half an hour, similar to a distant rustling, then gradually disappearing, and generally not re-appearing during the day. The examination with the constant current, with six Siemens-Halske's elements, showed, at the closure of the cathode, a very high singing noise, which continued only a short time during the duration of the cathode. On the closure and duration of the anode there was no reaction, but a short singing on the opening of the anode.*

The operation had no influence on the hearing-power of the left ear.

On the 29th December I repeated all the experiments, with the same results. The same again three times in the first week of January.

On a minute review of the described changes and the

* An electrode was held in the hand, and a metallic staff covered with flannel in the funnel-shaped cicatrix of the obliterated meatus, filled with salt water.

results of the functional examination, the greatest analogy is seen between this and the former case. The injuries of the external ears were nearly the same, except that the direction of the bullet was reversed. What changes in the drum-head and in the middle ear may have been present during the progress of the disease was very difficult to determine afterwards. At the time of the examination it is hardly possible that important changes were still present in this region.

I consider it, however, very likely, that in this case the primary lesion was likewise a change in the whole labyrinth, probably a similar effusion of blood, resulting from the injury, *i. e.* the concussion. The secondary changes superinduced by the same causes led to so-called nervous deafness. It is hardly possible to conceive of a central cause for this disturbance of function, as the affection was only on one side, and no brain symptoms whatever were present. The results obtained by the experiments with the tuning-fork, which agree with those of the first case, strengthen this diagnosis. Had in this case the labyrinth been uninjured, then, on account of the changes in the external meatus, resonance must have appeared on the left or injured ear, which was not the case. There were later continuous subjective sensations of hearing.

Based on my examination I advised the patient not to resort to an operation, which would give him fresh trouble, but never any improvement or restoration of hearing.

CASE III.—*Gunshot wound in the cartilaginous right ear—Labyrinth free—Serum in the cavitas tympani.*

A soldier of the Thirty-fourth Prussian Infantry was brought to me Feb. 3d by Dr. Lossen. He was wounded Jan. 18th, in the fighting on the Lisaine. The ball struck the right ear and passed below the left side of the lower jaw and came out. The consequences were so unimportant that the patient could be brought to me for examination without trouble. The examination showed a small slit-shaped wound of the lower wall of the cartilaginous meatus, 3‴ long and 1½‴ broad, but little suppuration, and upon probing as well as examination with the mirror the osseous meatus uninjured. The membrana tympani had that peculiar greenish-yellow coloration which is characteristic of serous accumulation in the tympanum, without, however, any line of demarcation whatever. Upon introduction of the catheter the turning of the instrument occasioned some pain. Auscultation during inflation made the impression as if air were blown into a vessel filled with fluid. This phenomenon is common in serous accumulations in the tympanum. The acuteness of hearing, which was before 1′ for a watch, was now increased to 3′, and he felt much relieved on that side. Bone conduction present. The tuning-fork was heard on the temple of the right side. No subjective noises. He was asked to come back for a paracentesis, but did not return.

In this case, where only the cartilaginous meatus was injured, the labyrinth remained free. The affection of the tympanum I looked upon as an accidental complication which had nothing to do with the injury.

CASE IV.—*Alleged sudden total deafness on the right side during battle—Suppurative catarrh of the tympanum, with perforation of the membrane—Adhesion of the latter to the wall of the labyrinth—Anchylosis of the stirrup.*

Prof. Julius Arnold sent me the right petrous bone of a soldier who was presumed to have become totally deaf during battle. Although

no particulars whatever with regard to the antecedents of the man or his disease could be obtained, I consider it neverthless desirable to publish the case on account of the appearance of the stirrup, in expectation of later similar observations.

Post-mortem appearances of the petrous bone. A great deal of pus in the external auditory meatus. The integumental lining thickened. Membrana tympani grayish-red, thickened, cloudy, anteriorly and superiorly, perforation the size of a pin's head, the rest almost totally united to the wall of the labyrinth. The mucous membrane of the tympanum thickened, and very red; as well as the cells of the mastoid processes, which were also full of pus. *The foot-plate of the stirrup* (dissected from the vestibule) *totally immovable.* No changes in the vestibule and the cochlea.

The laceration of the membrana tympani and the consecutive suppurative catarrh of the tympanum after a violent cannonade are not at all surprising. The adhesion of the membrana tympani to the wall of the labyrinth was probably caused by the one-sided action of the tensor tympani. The synostosis of the stirrup, however, was exceedingly remarkable, for if its development dates only from the time of the battle, the appearances would throw a peculiar light upon the pathological anatomy of many cases of severe deafness occasioned by cannonading. In many such cases, an almost acute development of stapedial synostosis might take place. One may, of course, and rightly, object that there is nothing known of the antecedents; the whole affection may possibly be of older date, and perhaps aggravated by the cannonade. To refute this objection, it may be said that a person with such alterations in the organs of hearing, necessitating al-

most complete deafness, would hardly have been considered fit for military duty. Does the mechanical irritation of the stapedial region, which must follow such lacerations of the membrana tympani, and attain a high degree if the cannonading continues after the occurrence of the perforation—does this irritation, combined with the inflammation of the membrana tympani adherent to the promontory, lead to the rapid formation of a synostosis of the stapes? I do not dare to answer this question, but cannot refrain from directing the attention of the profession to this point. In literature are found several cases of immobility of the stapes from gun or cannon shots, but in all cases a longer time had elapsed before a high degree of deafness and anchylosis ensued, and in accordance with this the autopsies were made only many years afterwards; in one case the immobility of the stapes was produced by surrounding adhesive bands. (Compare Toynbee's Catalogue, No. 567 and 68, 577 and 78, 597 and 98.) I have nowhere found descriptions of post-mortem conditions similar to ours, with sudden development of anchylosis of the stapes, and future observations must show whether or not such anchylosis can be produced by cannonading and similar external causes.

CASE OF CONTRACTION OF THE LEFT TENSOR TYMPANI SYNCHRONOUS WITH THE ACT OF MASTICATION.

BY DR. MOOS.

Translated by J. H. Pooley, M.D., of Yonkers, N. Y.

MADAME G., 37 years old, consulted me regarding a double-sided trouble of the ears, of four years' duration. On the left side, she says, that as long as she can remember she suffers with a loud grating, which, however, is only present during mastication. While eating, her hearing on the left side, she says, is worse, and a strong sensation of pressure is felt in the left ear.

The examination of the function of hearing showed a difference in the acuteness of hearing; the right side 37″, left side 12″, for a watch of 30′ hearing distance. Bone conduction for the watch and tuning-fork absent; the latter is only perceptible on the left side; catarrh of the pharynx, both tubes, and tympanum. After treatment for three weeks the hearing distance on the right side 9′, left side 2½′. The treatment had no influence on the grating sound. This was heard very plainly without any instrument, near the patient, on movements of the lower jaw. The examination of both membranæ tympanorum showed no material anomaly; on examination, while the patient was chewing, the left membrana tympani showed a to and fro movement. The light spot became smaller during the drawing in of the membrane. The sound of tuning forks C′ and C″, vibrating on the cranial bones, more intense during the experiment, but enfeebled when held before the ear. On the right side I was not able to observe anything similar during chewing, upon examination of the membrana tympani, nor had the patient any similar sensation on the right side as on the left.

The phenomenon is especially remarkable because the result was confined to one side. If it had been observed on both sides it would have been less remarkable, as the muscles used in chewing and the tensor tympani receive their nerves from the same source. (On voluntary contraction of both tensores tympani, compare A. Schapringer. Transactions Austrian Acad. of Sciences, Vol. 62, Sect. 2, Oct. No. 1870. Presented Oct. 13th, 1870.) As is well known, Helmholtz first drew attention to the grating noise caused by violent concussions of the ear. He explains it by the striking together of the ratchet-teeth of the malleo-incudal joint. Politzer contests the correctness of this explanation from experiments of his own. According to him, the sound is derived from the whizzing noise of the membrane and the ligaments of the ossicula. Politzer could produce this whizzing in the ear of a corpse, even when the malleo-incudal joint was artificially anchylosed. (Wochenblatt der k.k. Gesellschaft der Aerzte, No. 15, Vienna, 1870.)

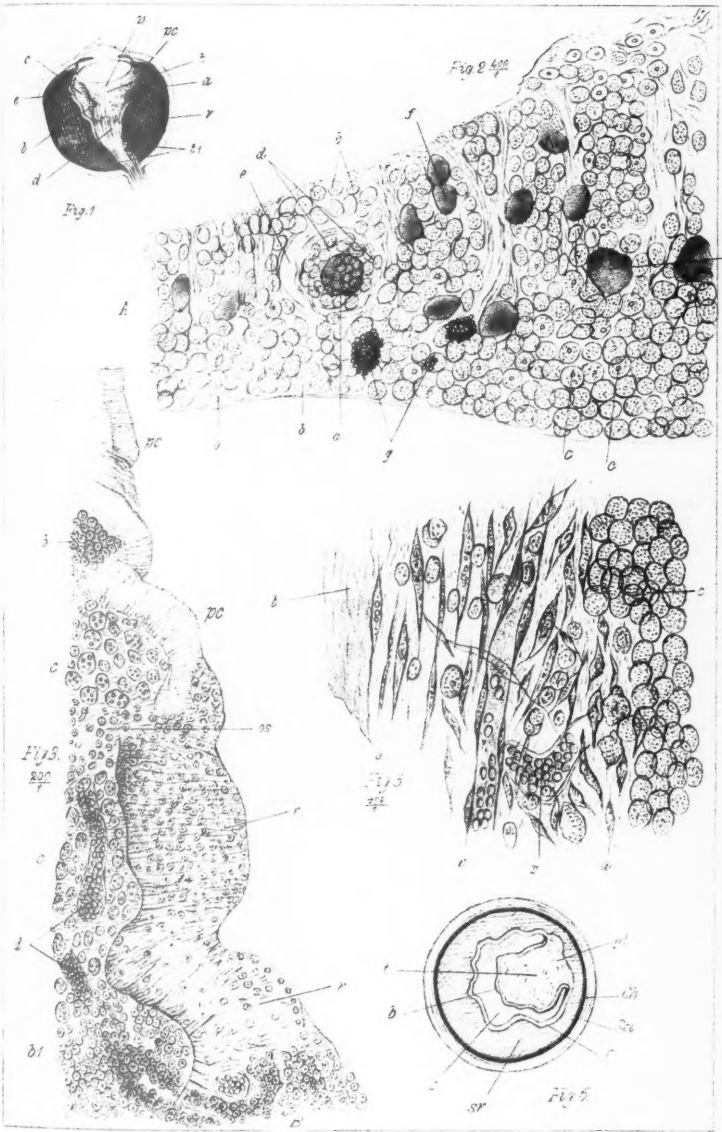
Fig. 1
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Fig. 3. 200/1
Fig. 4 225/1
Fig. 5.
H. Knapp ad nat. del.
A. Mutzenbecher Lith.

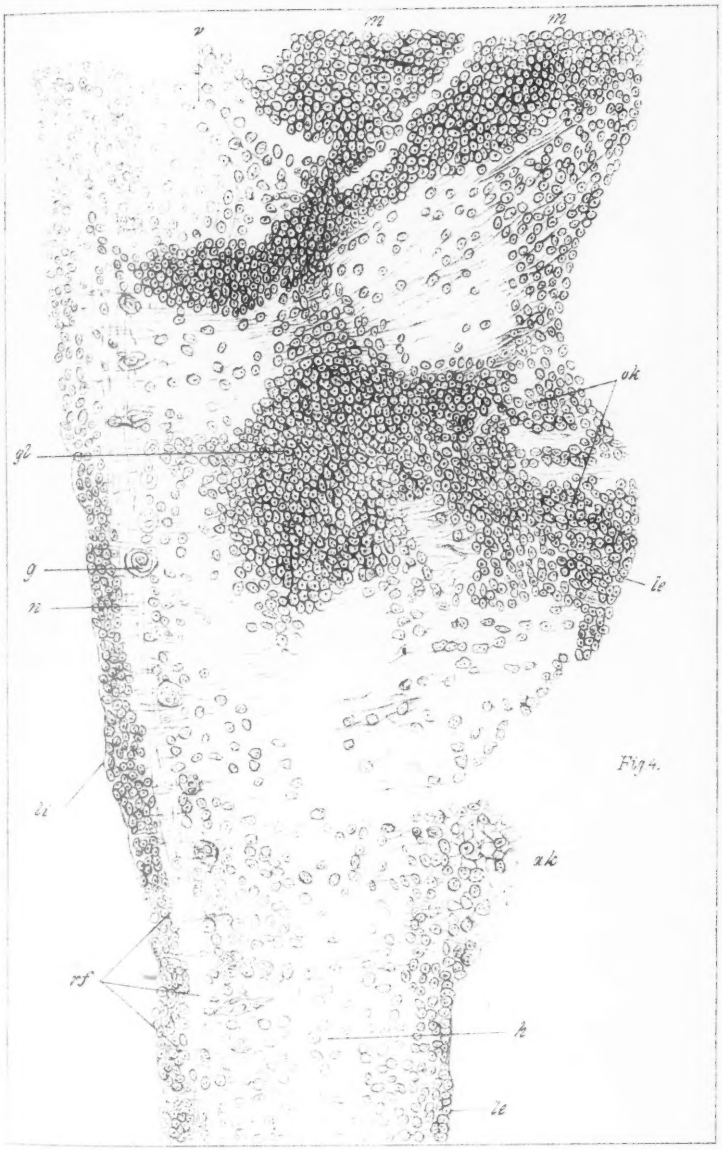

Fig. 4.

H. Knapp ad nat. del. A. Meisenbach Riffarth & Co. Lith.

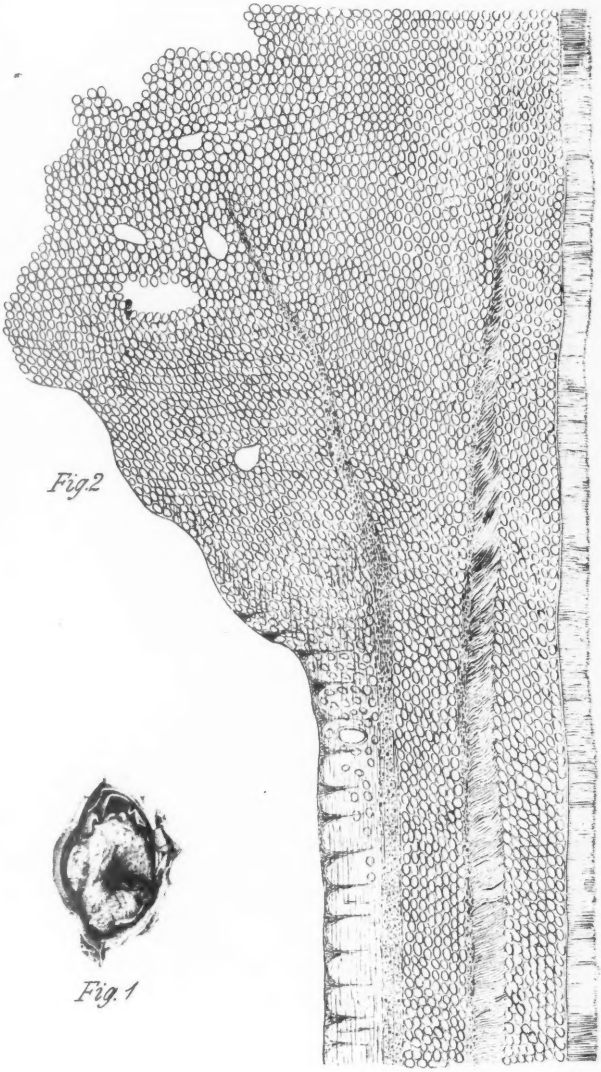

Fig. 2

Fig. 1

F. Delafield, praep.
H. Eno ad nat. del.

J. Maisonneuve Lith.

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Fig. 3.

Fig. 4.

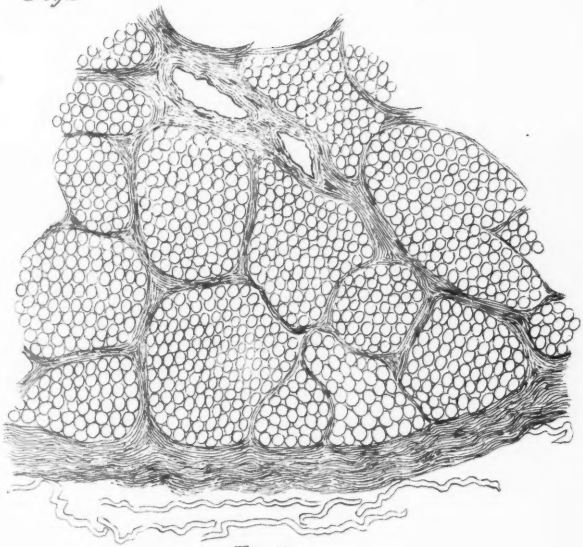


Fig. 5

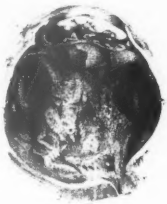
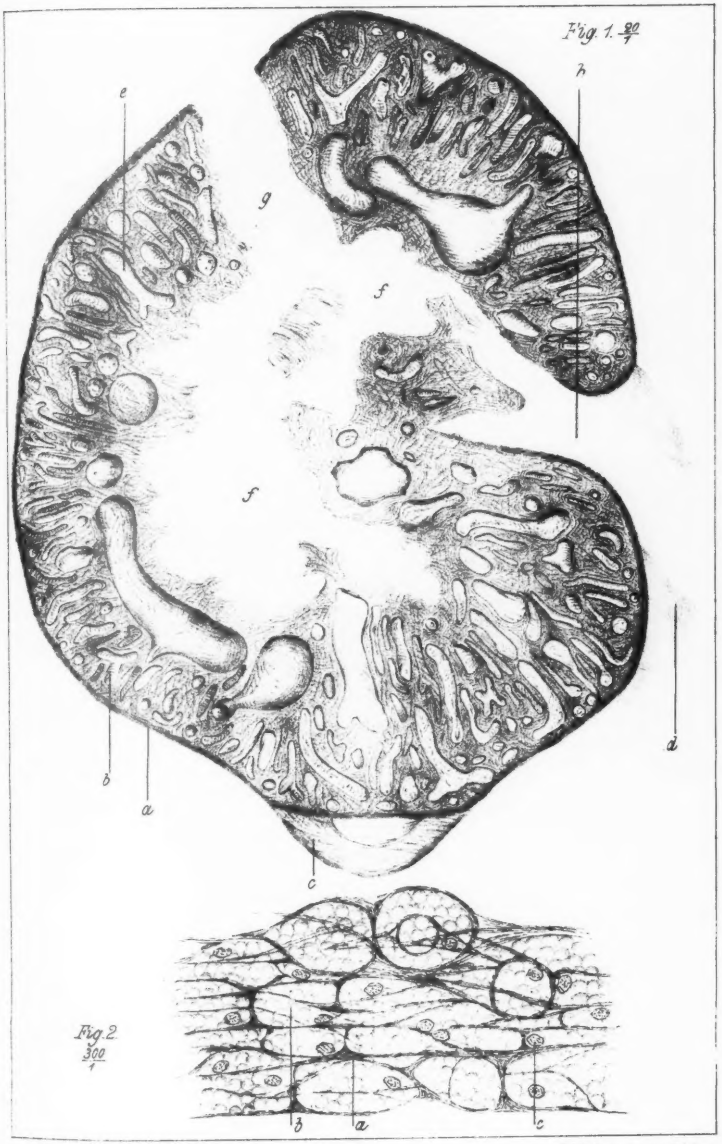


Fig. 6.

Fig. 7.

F. Delafield praep. J. Maisonneuve Lith.

Tab. VI.



A.H.Buck ad nat. del. J.Maisonneuve Lith.

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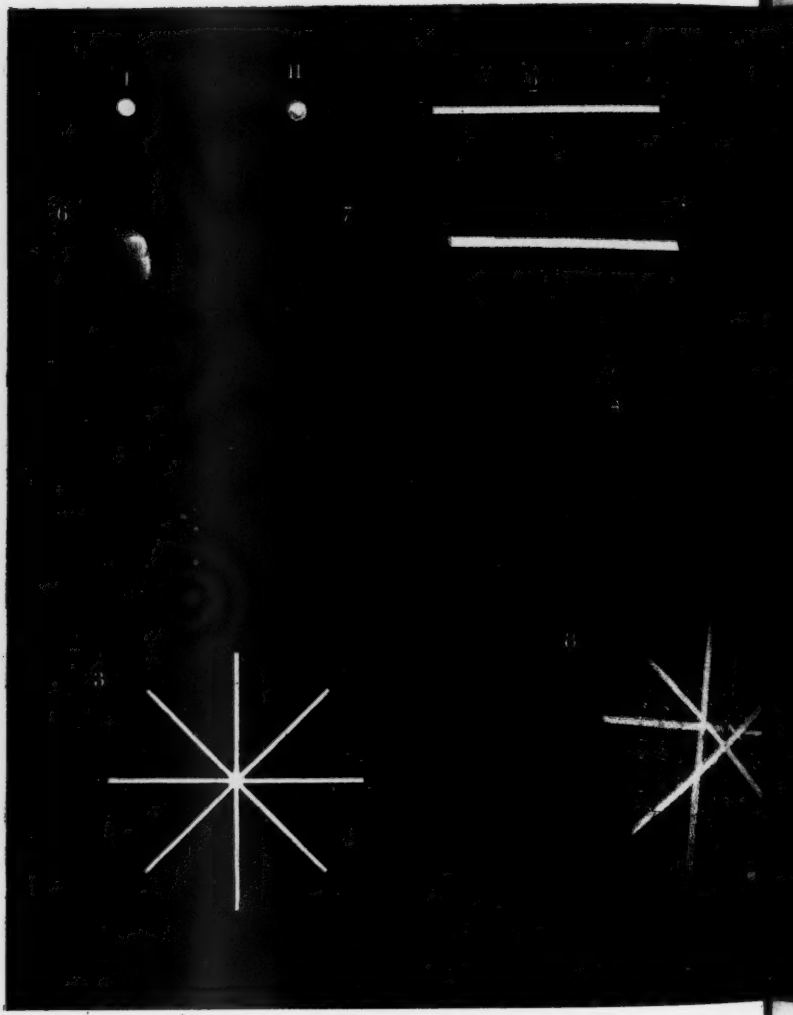

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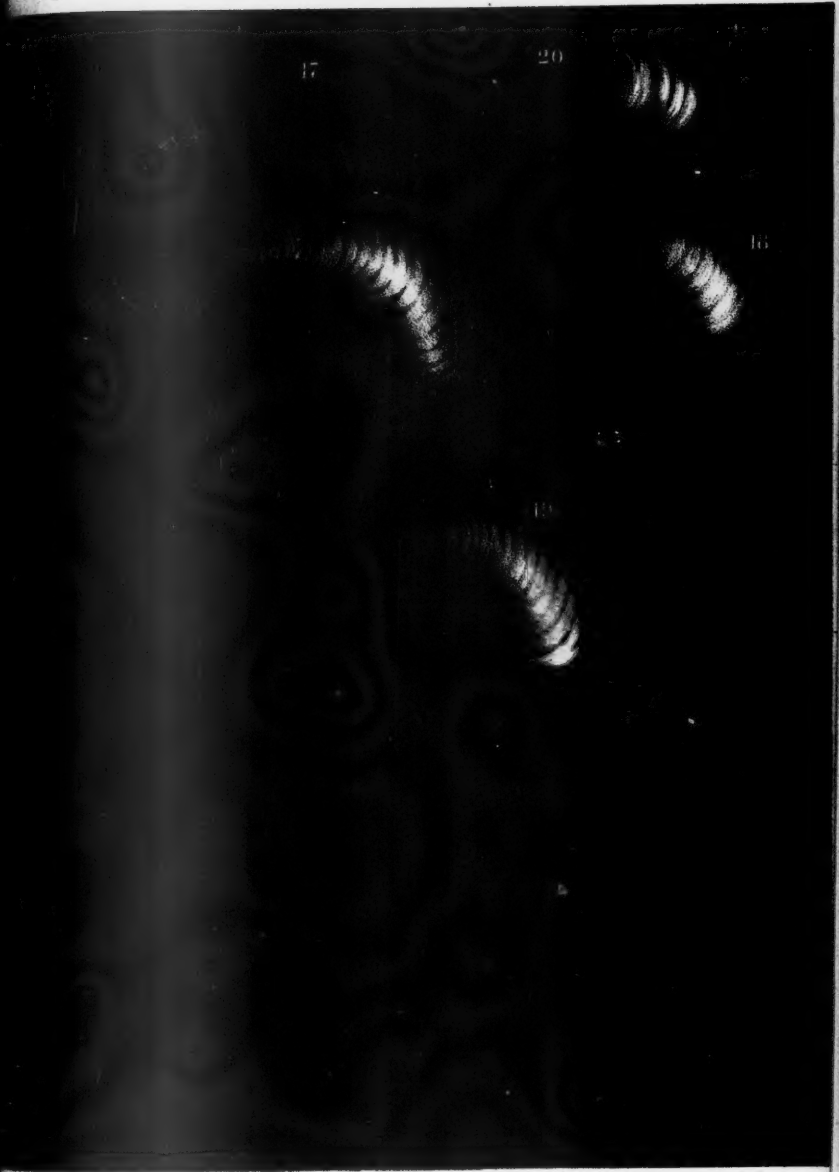

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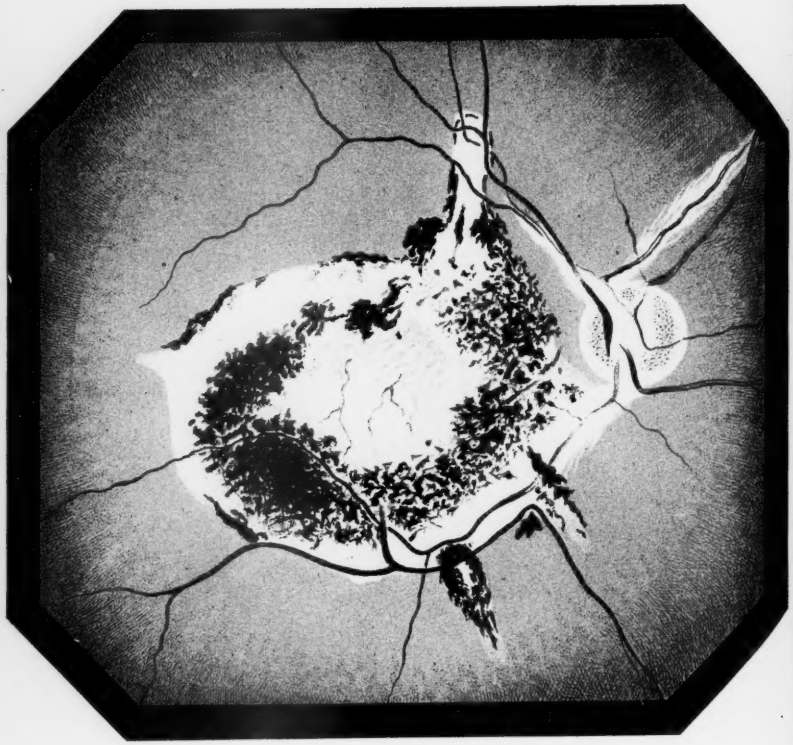

Fig. 1.

Dr. E Loring ad. nat.

Carlsruhe. Chr Fr Müller'sche Lith. Anstalt

Knapp & Moos, Archiv II, 2.